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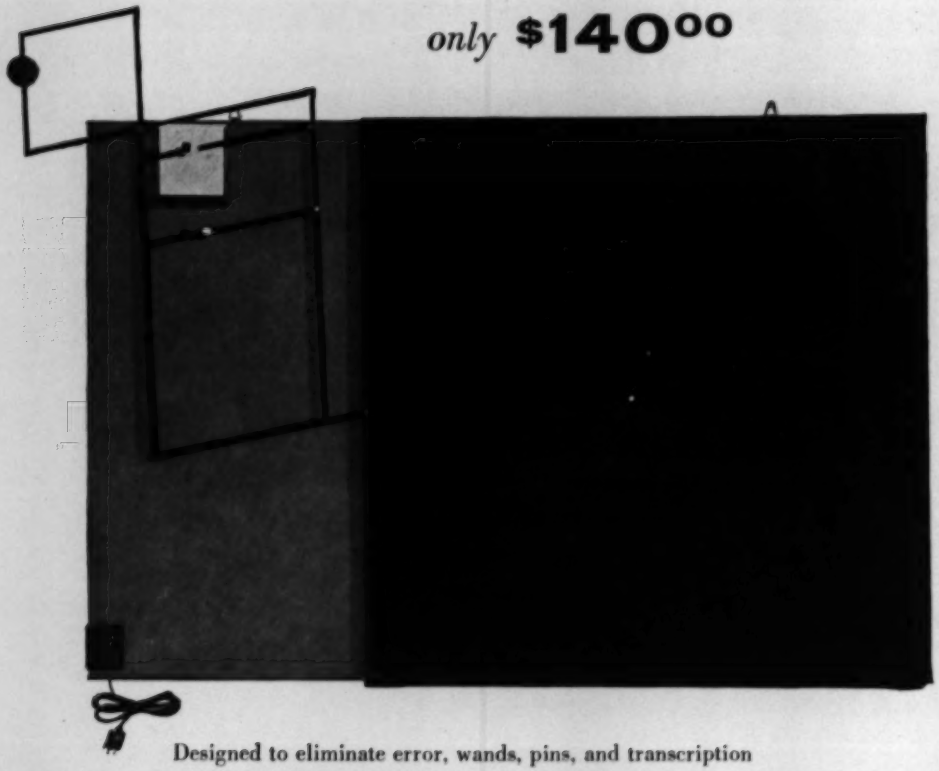

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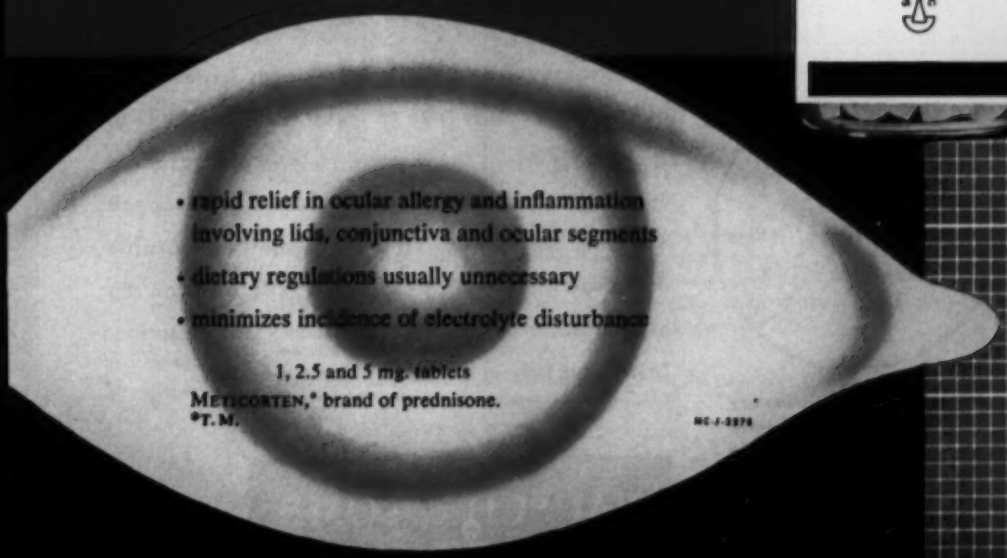

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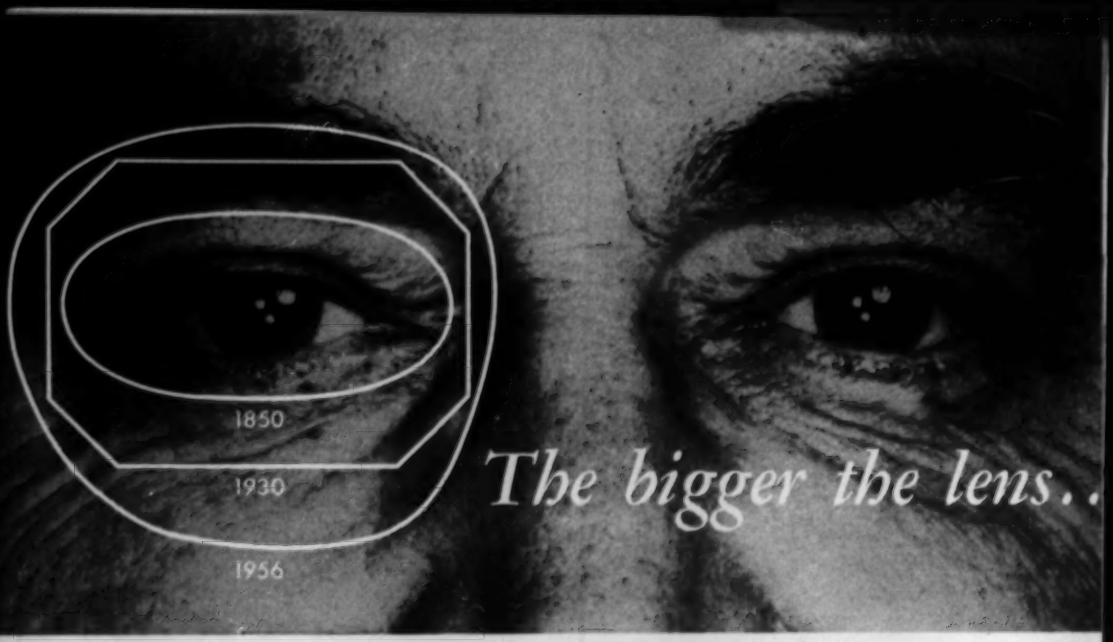

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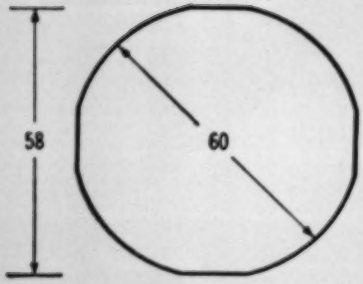

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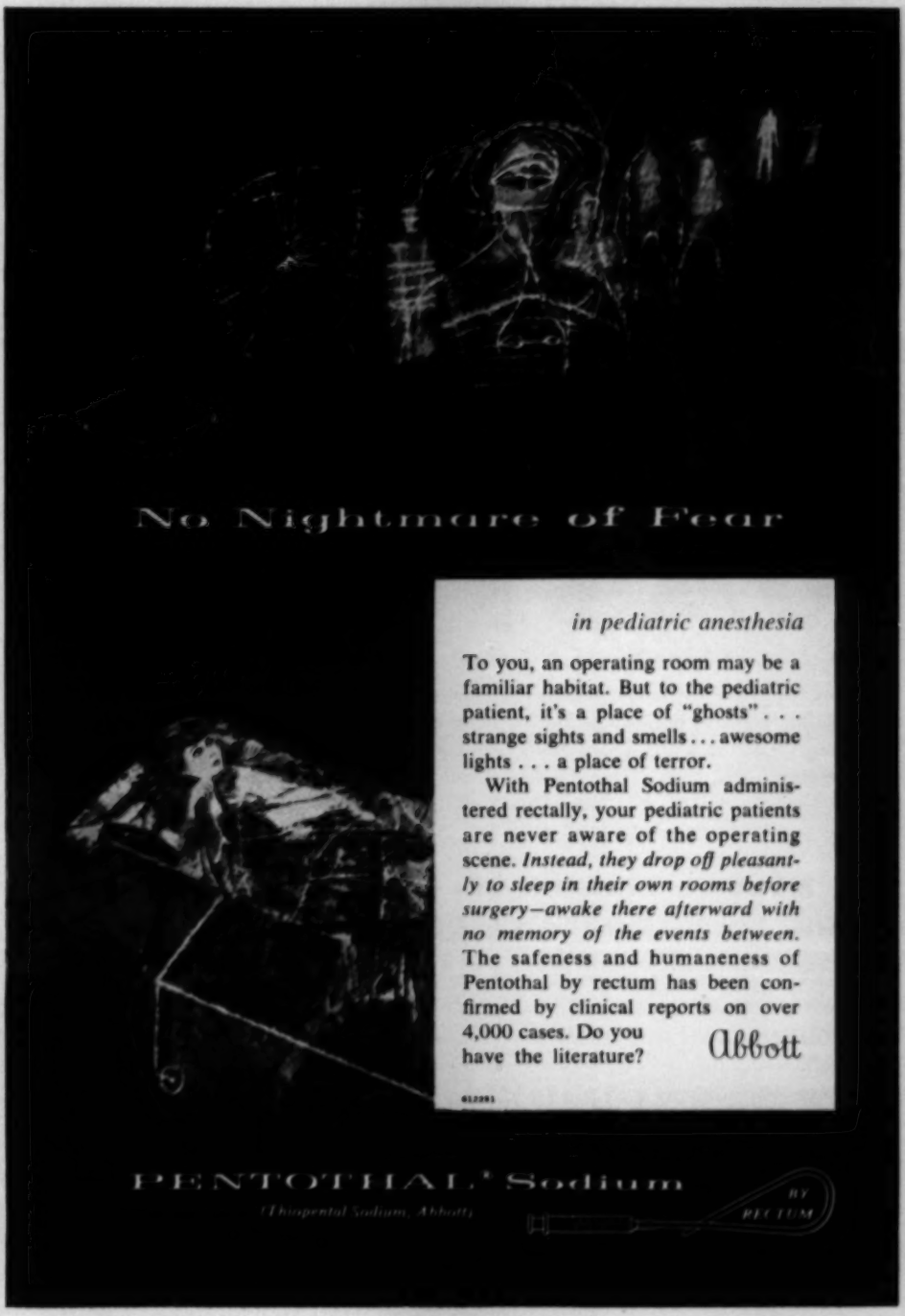

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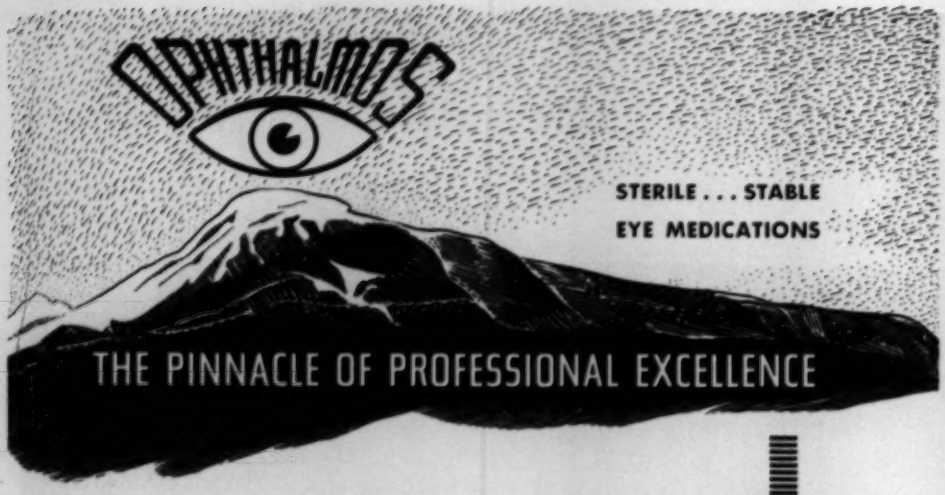

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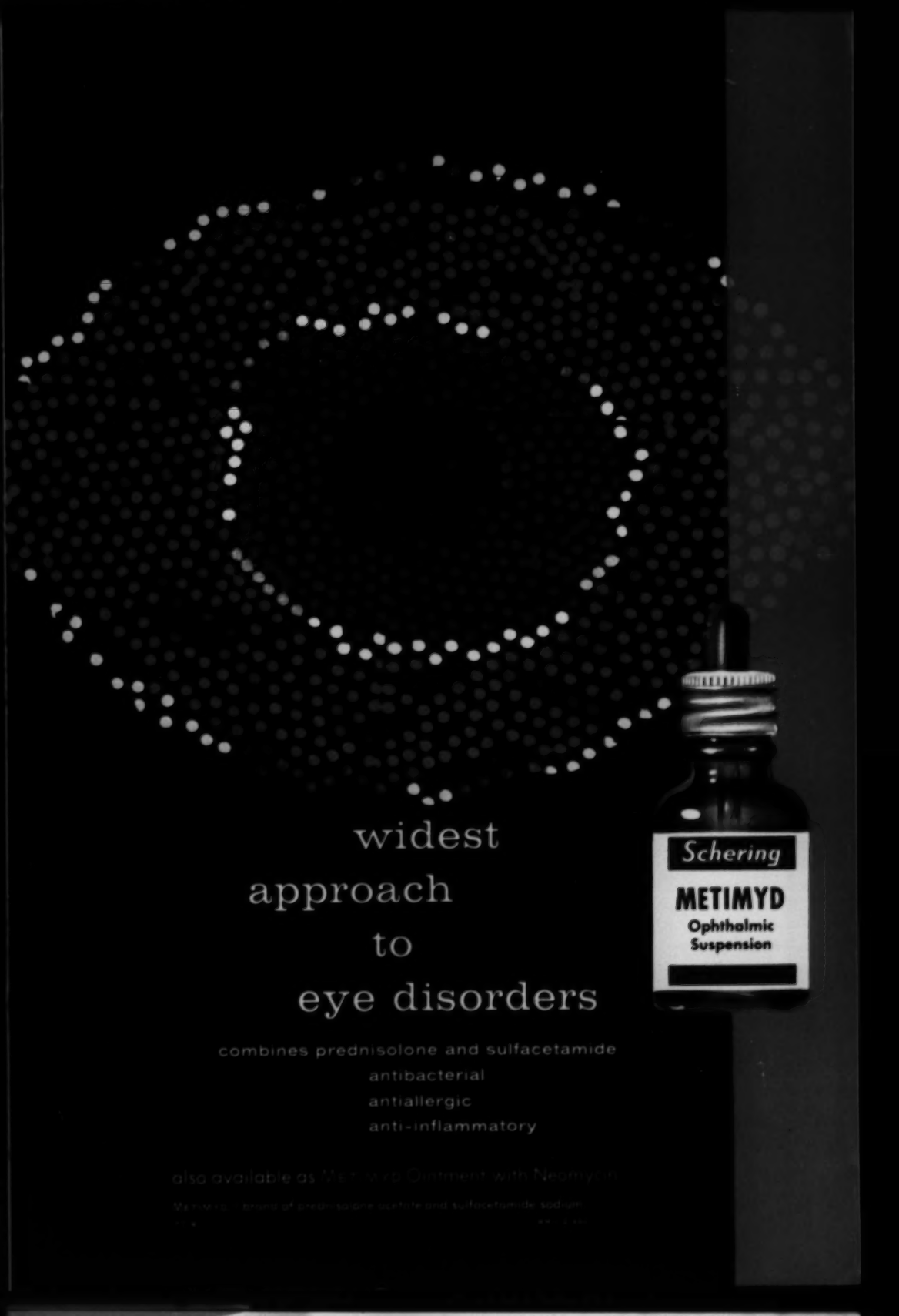
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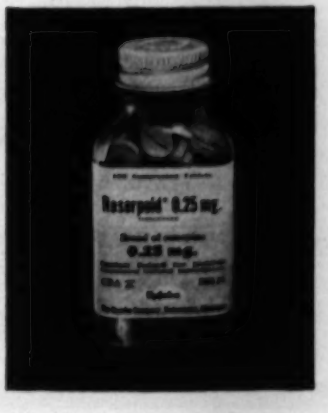

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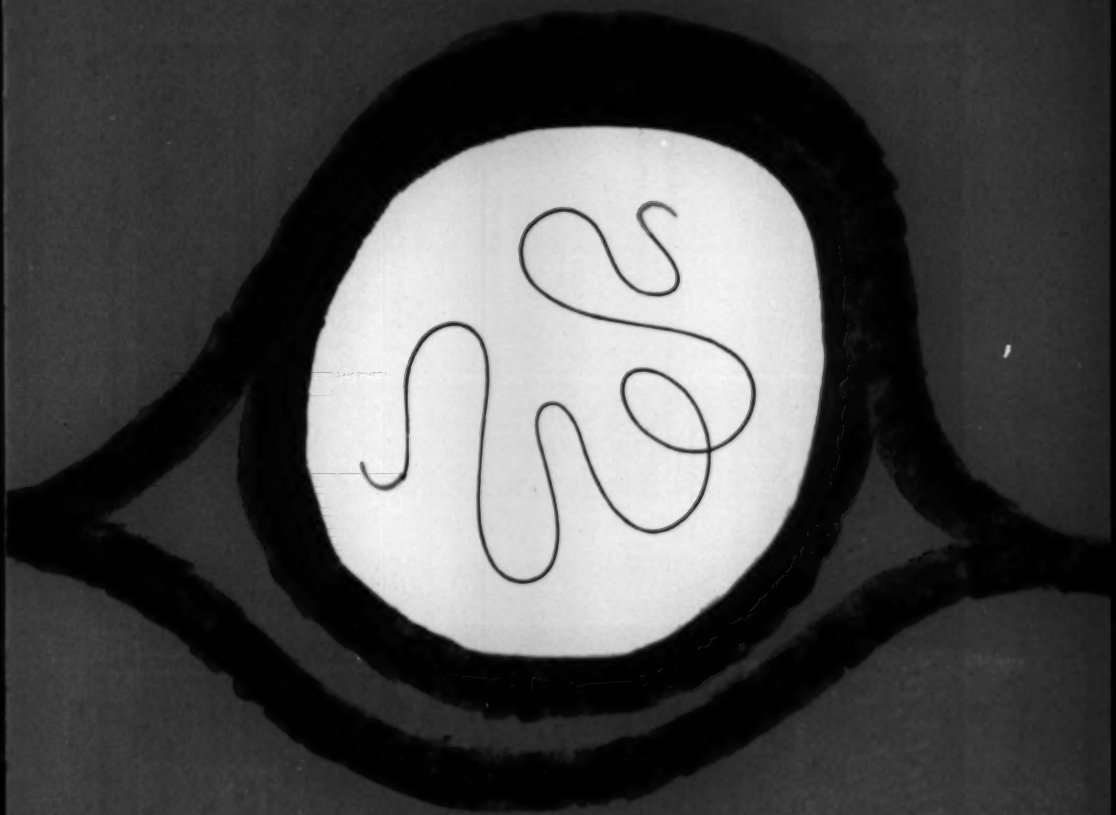

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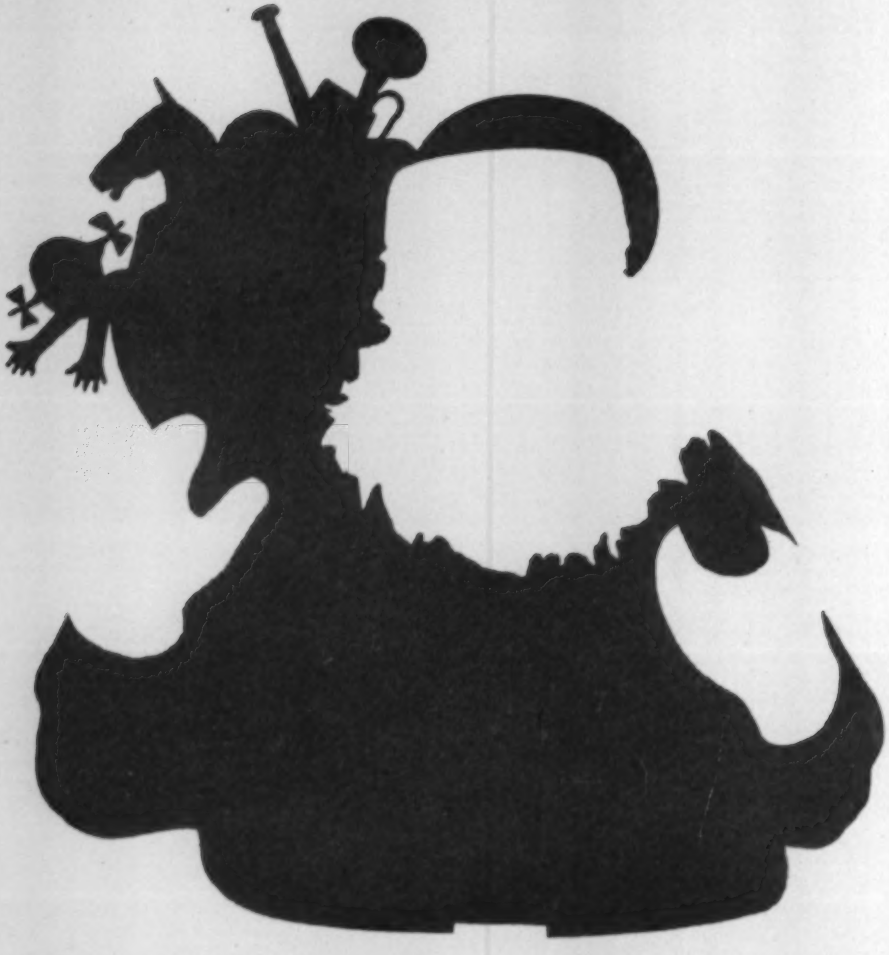

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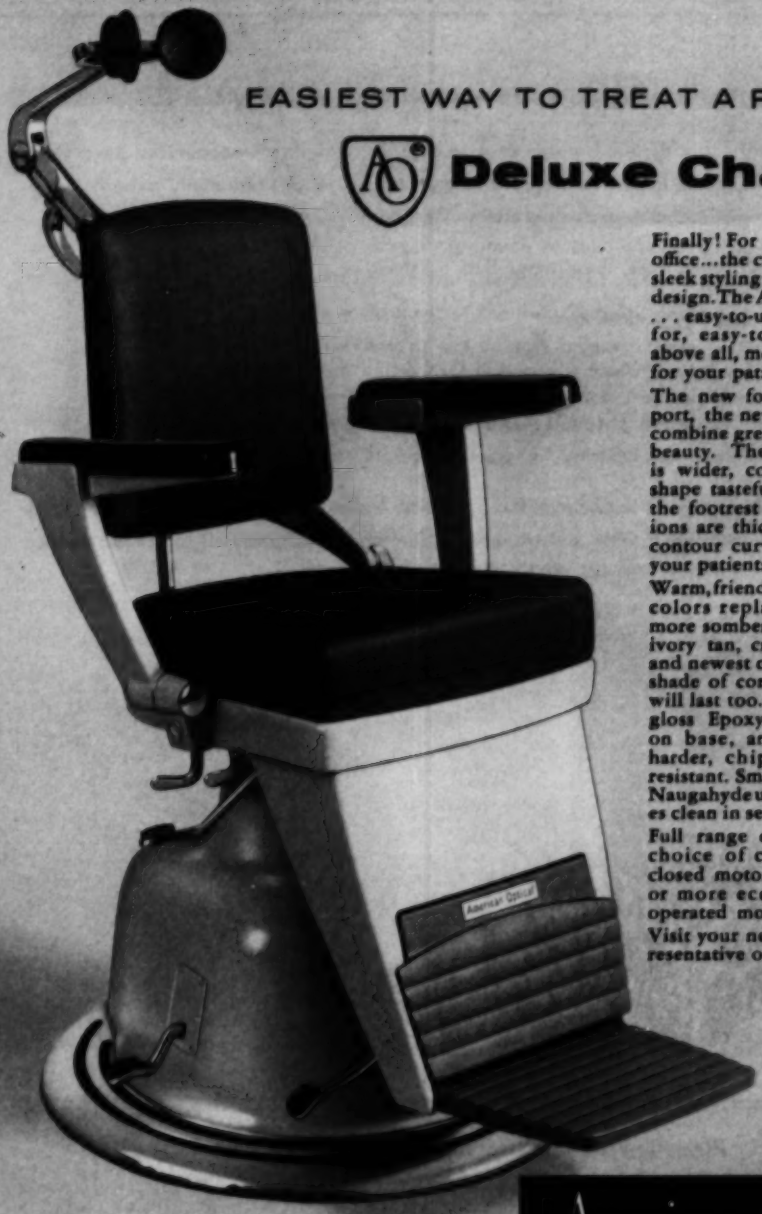

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1. Rasgorshek, R. H., and McIntire, W. C.: Am. J. Ophth. *40*:34 (July) 1955.
2. Council on Pharmacy and Chemistry, A.M.A.: New and Nonofficial Remedies, Philadelphia, J. B. Lippincott Company, 1955, p. 263.
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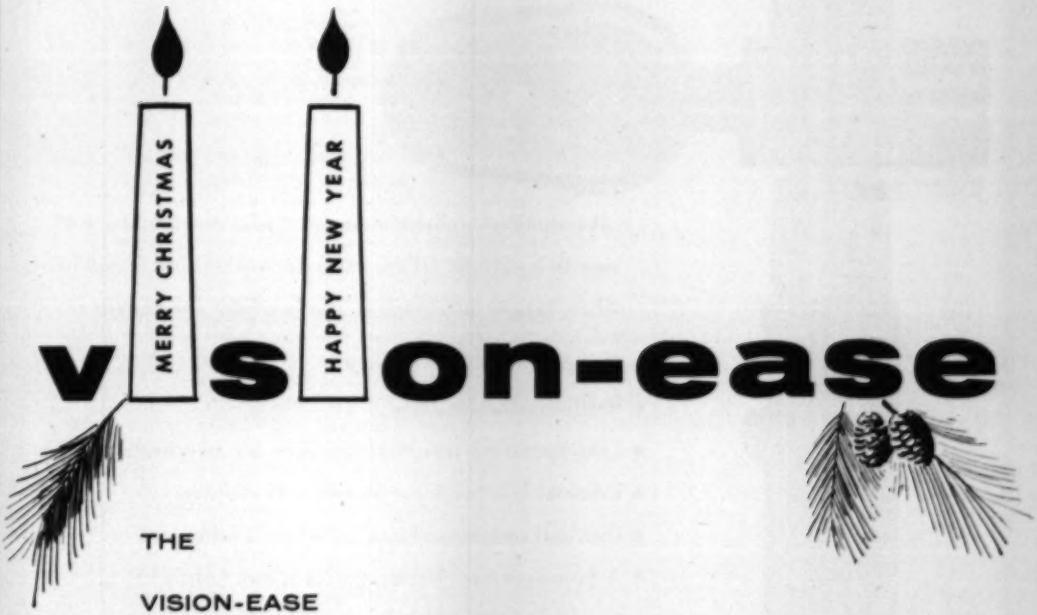

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ABSTRACTS

Anatomy, embryology, and comparative ophthalmology; General pathology, bacteriology, immunology; Vegetative physiology, biochemistry, pharmacology, toxicology; Physiologic optics, refraction, color vision; Diagnosis and therapy; Ocular motility; Conjunctiva, cornea, sclera; Uvea, sympathetic disease, aqueous; Glaucoma and ocular tension; Crystalline lens; Retina and vitreous; Neuro-ophthalmology; Eyeball, orbit, sinuses; Eyelids, lacrimal apparatus; Injuries; Systemic disease and parasites 943

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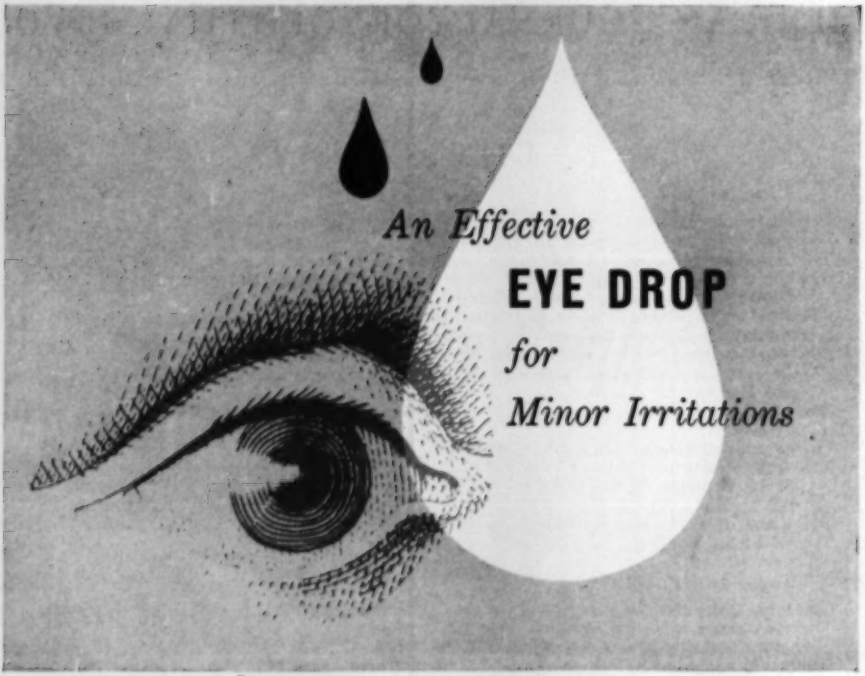

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VOLUME 42 DECEMBER, 1956 NUMBER 6

RETINAL LESIONS ASSOCIATED WITH UVEAL DISEASE: PART I*

BERTHA A. KLIEN, M.D.
Chicago, Illinois

Recognition of involvement of the retina in the course of an anterior or posterior uveitis is of importance for several reasons, *firstly*, it may explain an otherwise puzzling diminution or loss of vision out of proportion to the degree of the uveal disease; *secondly*, it may prevent incorrect diagnosis and treatment in those cases in which the initial subjective symptoms are overwhelmingly due to the retinal participation in the disease process; and *thirdly*, certain retinal phenomena, especially if searched for and observed systematically in large groups of patients with uveitis, may eventually aid in the evaluation of severity and prognosis of the uveal disease in certain cases.

The retinal lesions associated with uveal disease to be discussed are: retinitis serosa, arteriolitis, periphlebitis, capillaritis, central angiospastic retinopathy, and retinal separation.

I. RETINITIS SEROSA

The first part of the term, retinitis serosa, correctly implies the inflammatory nature of the disease; the second, somewhat meaningless part emphasizes its similarity to iritis serosa, a nonspecific infiltrating type of inflammation devoid of marked exudation but associated with formation of precipitates. Samuels,[1] who described in detail the histopathologic findings in the related retinal condition, retained the term, serosa, otherwise it might be described as a low-grade, nonspecific, infiltrating retinitis. It occurs in postoperative or posttraumatic penetration of the eyeball accompanying a low-grade, nonspecific infiltrating iritis, but occasionally accompanies the late stages of a chronic endogenous infiltrating iritis or iridocyclitis.

CASE 1

S. A., a man 75 years of age, with bilateral nuclear cataracts, entered Billings Memorial Hospital for extraction of the right lens. An intracapsular extraction was intended but the capsule ruptured and a small amount of cortex remained in the anterior chamber.

These remnants absorbed rapidly but the eye remained injected for six weeks, although corrected vision after five weeks was 20/30 and a temporary lens was ordered.

The fundus at that time was clearly visible and appeared normal, and no cause for the slightly reduced vision could be found.

Five months postoperatively the patient returned to have another refraction prior to the order of the permanent lens. He stated that for the past two weeks the vision in the operated eye was not quite as good as before.

The best corrected vision obtainable at that time was 20/40−3.

The right globe was pale; there was diffuse pigment dispersion over the posterior corneal surface and over the anterior surface of the slightly herniated vitreous.

There were a few formed floaters in the vitreous but the fundus was still clearly

* From the University of Chicago, Foundation for Ophthalmic Research. Read before the Clinical Conference of the Chicago Ophthalmological Society, February, 1956.

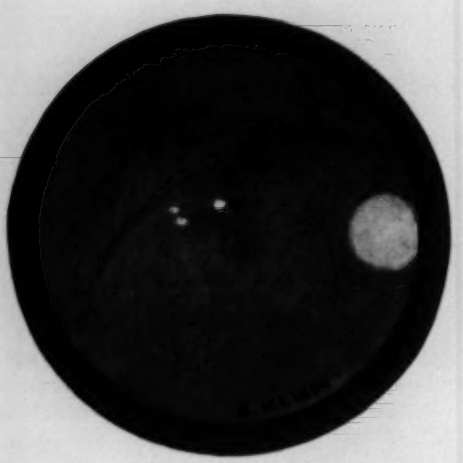

Fig. 1 (Klien). *Case 1.* A man, 75 years of age, lens extraction four and one-half months ago. Incipient detachment of vitreous with two postvitreal precipitates.

visible. Optic disc and retinal vessels appeared to be normal. In the upper macular region there were two faint semicircular lines suggesting an incipient detachment of the vitreous (fig. 1) which was verified by observation with the Gullstrand ophthalmoscope. Several pinpoint-sized white dots just above the fovea centralis appeared to be in front of the retinal level and gave the impression of being attached to the posterior surface of the detached vitreous. The foveolar reflex was absent.

A month after these changes were first observed, the vision of this eye was still 20/40−3, but the vitreous was completely detached, lying in the anterior half of the cavum oculi. The posterior surface of the vitreous showed two spiderlike opacities, with dense white centers, which seemed to have resulted from contraction of this surface around two small white deposits. They were possibly the same ones which were observed previously.

CASE 2

T. H., a man, aged 71 years, sustained a penetrating injury of his left cornea with a piece of flying glass four months prior to the time of enucleation of this eye.

The patient was first seen and hospitalized in the Presbyterian Hospital of Chicago three days after the injury. An extensive y-shaped cut in the cornea was found with incarceration of iris into the nasal limb. The eye was treated conservatively with heat and atropine, since these events took place prior to the advent of antibiotics.

Three weeks after the injury a secondary glaucoma developed and was treated with a basal iridectomy which normalized the tension. The globe gradually lost its intense mixed injection and became painless although never completely pale.

One week before enucleation pain recurred. The eye was found intensely injected with a dense adherent leukoma in the center of the cornea. The iris pattern was indistinct and the lens was opaque. Tactile tension was soft. The vision was only light perception and the eye was removed because of the danger of sympathetic ophthalmia.

Histologic examination of the globe revealed incarceration into the penetrating scar not only of iris but also of lens capsule and some lens cortex.

There were no polymorphs or giant cells around the lens remnants. Iris and ciliary body were densely infiltrated with lymphocytes and plasma cells, and small precipitates composed of these cells lined the anterior surface of the iris and the free surface of the trabecula corneosclerale. In several places there were short peripheral anterior synechias.

Precipitates, consisting of lymphocytes and some mononuclear leukocytes, lined the inner surface of the retina (fig. 2) and the posterior surface of the extensively detached vitreous (fig. 3).

In addition to the precipitates there were many elongated fibroblastlike cells growing over the posterior vitreal surface, joining up with one another in most places, thus forming a thin membrane over this surface. The vitreous itself contained scattered

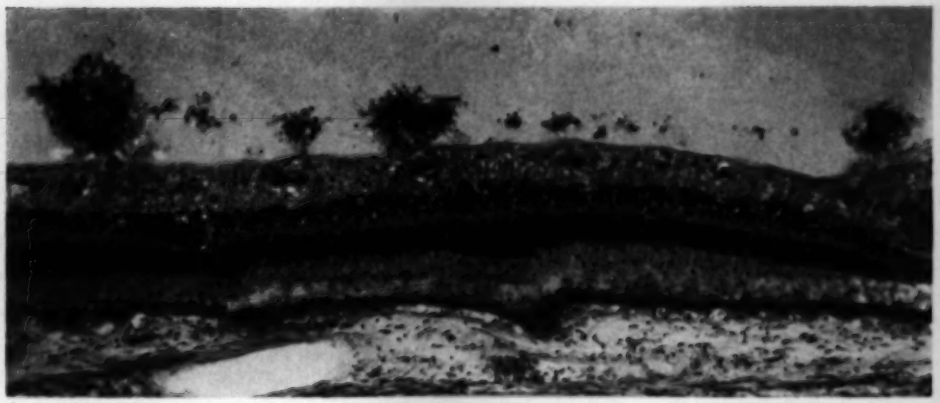

Fig. 2 (Klien). *Case 2.* A man, 71 years of age, penetrating injury four months ago, retinitis serosa. Inner surface of retina lined by round conglomerations of lymphocytes and monocytes: preretinal precipitates.

lymphocytes, either as single cells or as small conglomerations.

In the macular region rows of plasma cells had accumulated underneath the internal limiting membrane (fig. 4) and there was some perivenous and pericapillary cuffing with lymphocytes and plasma cells. The nerve fiber layer was thickened and edematous, while the outer retinal layers were normal.

The optic papilla was edematous and there was a peripapillary circumscribed retinal detachment, findings produced in this case by low intraocular pressure.

A diagnosis of an exacerbation of a chronic posttraumatic, nonspecific infiltrating iridocyclitis and retinitis was made. A lens-induced inflammation, in the sense of a toxic not a sensitivity reaction (Irvine, S. R.[2]) was considered a possibility.

Fig. 3 (Klien). *Case 2.* Extensive posterior detachment of vitreous (v). Postvitreal precipitates similar to preretinal precipitates, posterior surface of vitreous lined with fibroblasts which join one another to form thin membrane.

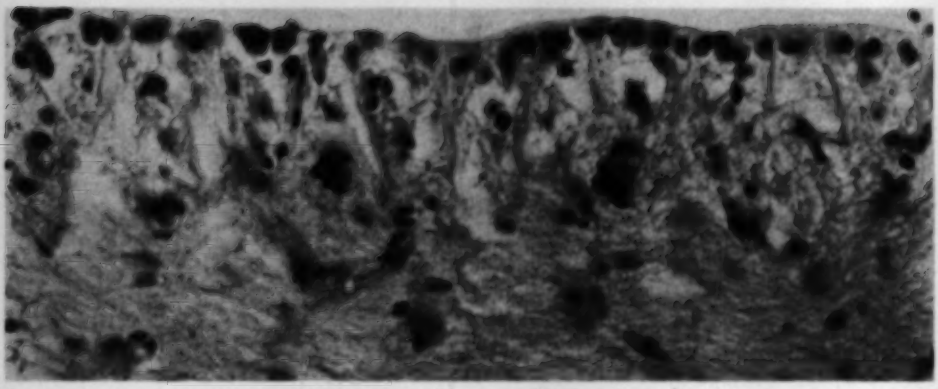

Fig. 4 (Klien). *Case 2.* Rows of plasma cells underneath internal limiting membrane, slightly reducing transparency of retina.

Discussion of Cases 1 and 2

In discussing the clinically observed and the histologically studied case together one might underscore and correlate certain subjective and objective phenomena and be able to circumscribe more definitely the clinical picture of retinitis serosa.

The infiltration with round cells of the inner layers of the retina accompanied by edematous thickening of the nerve-fiber layer, which characterizes the histopathologic picture must surely escape clinical observation unless these changes are pronounced in the perifoveal region. In that case they produce irregularities of the clivus and a visible distortion and perhaps disappearance of the foveal reflex. Irregularities of the macular region in general of similar origin may be observable in younger individuals through distortion of the macular reflex.

Infiltration and edema of the retina, even if limited to its innermost layers, must cause a moderate decrease of its transparency and thus explain the slight diminution of central vision so characteristic of these cases.

This diminution of vision is not associated with metamorphopsia or with a positive central scotoma, at least not as a rule. In this respect the serous retinitis differs from the serous retinopathy (central angiospatic retinopathy). The abscence of metamorphopsia may be explained by the limitation of the edema to the inner layers of the retina, the absence of a central positive scotoma by the fact that no detachment of the internal limiting membrane, such as characterizes the functional vascular disease, seems to develop in the inflammatory condition.

The entire disease process in retinitis serosa is low grade and chronic and need not remain confined to the retina as we know from the histologic and suspect from the clinical picture.

Extraretinal inflammatory cells, namely, lymphocytes and monocytes, may wander into the vitreous or remain between retina and vitreous. The latter are gradually transformed into fibroblasts forming a thin membrane on the posterior vitreal surface. Such membrane formation has several consequences: shrinkage of the membrane leads to progressive detachment of the vitreous, and its mere presence must reduce the permeability of the posterior vitreal surface, thus leading to retention of more and more cells behind it in the form of postvitreal or preretinal precipitates.

We have no knowledge of the earliest stages of these events or of their exact se-

quence. Clinical observations, such as are demonstrated in Case 1, reveal that postvitreal precipitates may be clinically visible when the vitreous detachment is very low and apparently just beginning. The onset of the vitreous detachment in this patient, who had a visual acuity of only 20/200 in the other eye, probably coincided with the time when he noted a slight diminution of vision, which occurred two weeks prior to our re-examination and four and one-half months after the lens extraction.

The prolonged if slight ciliary injection of the globe indicated a low grade postoperative anterior uveitis, again with the possibility of being lens induced. The participation of the retina, which could merely be suspected at the time when the corrected vision was subnormal without demonstrable cause, became more obvious upon discovery of postvitreal precipitates.

The onset of the vitreal detachment, four and one-half months after the operation without any sign of traction upon the vitreous arising from the anterior segment, strongly suggested the formation of a membrane on the posterior surface of the vitreous during this interval, with the above-mentioned consequences of decreased permeability and shortening of this surface. The rapidity with which the vitreal detachment became complete, and the visible foci of contraction on the posterior surface of the detached vitreous also supported this view.

There always exists the possibility that later in such a series of events membranes form also on the inner surface of the retina. Termed "preretinitis" by Leber, such membranes arise in places where contact between retina and vitreous had been lost for a long time. They may lead to foci of shrinkage of the inner retinal surface, to star folds, retinal holes, and tears, and to retinal separation.

The prognosis for an eye which has shown the sequence of events as in Case 1 is always guarded.

It would bear investigation to see what percentage of postoperative aphakic eyes which develop a retinal detachment had a prolonged recovery period and findings suggestive of retinitis serosa. The well known poorer surgical results in these eyes, especially with a surgical method which does not include scleral shortening, as compared to the results in retinal separation in non-aphakic eyes, could be accounted for by the existence of membranes on the inner retinal surface prior to the development of the retinal separation.

Summarizing briefly the at best subtle subjective and objective clinical findings in retinitis serosa, we see, nevertheless, a recognizable clinical picture before us. Its early uncomplicated stage is characterized by:

1. A slight diminution of central vision without central or paracentral scotomas and without metamorphopsia, not accounted for by observable pathologic findings.

2. Distortion of the foveal reflex and irregular reflexes in the macular area, such as may be produced by irregularities in the level of the internal limiting membrane due to cellular infiltration and edema just underneath it.

Its later stages complicated by:

3. Changes in the posterior vitreous, varying degrees of vitreal detachments, and perhaps more often than we realize, unless we look for them, postvitreal precipitates. Still later:

4. Membranes on the inner retinal surface with the inherent danger of retinal separation.

II. Retinal arteriolitis

In some cases of severe uveitis peculiar alterations of the retinal arterioles occur. All or isolated arteriolar branches show yellowish plaques which may vary in intensity of color or opaqueness. They appear as nodular thickenings around the blood column but actually are areas of opacity of the otherwise transparent vessel wall, never extending beyond the normal thickness of the wall in any size of arteriole. These

plaques never change their position, but gradually fade out as the uveal disease subsides, until after many months most or all of them have disappeared, leaving behind no visible alteration of the arteriolar walls.

CASE 3

B. W., a Negress, aged 23 years, came to the Illinois Eye and Ear Infirmary with the complaint of blurring of the right vision, and of floaters before the right eye of recent onset.

At the time of the first visit the right vision was 20/60. The left vision was 20/20 and the left eye was normal. There was fine corneal edema in the right eye with a tension of 53.0 mm. Hg (Schiøtz). An area of active chorioretinitis was discovered one disc diameter inferior and temporal to the disc.

Under atropine the tension of this eye became normal within one day. Fine precipitates over the inferior nasal quadrant of the cornea and several incipient posterior synechias were then noted.

Physical examination and laboratory tests were negative with the exception of a two plus reaction to the first strength of purified protein derivative (PPD). There also appeared to be a focal reaction to this diagnostic test in the form of increasing retinal edema around the chorioretinitic focus and an increasing number and size of the corneal precipitates. On the other hand, the disease was seen in its very beginning and the progression may have been the inherent unavoidable development and not a focal reaction. The visual acuity had diminished to 2/200.

Treatment consisted of streptomycin (0.5 gm. twice a week) and isoniacid (50 mg., four times daily) and later 100 mg. three times daily).

After three weeks, the right vision had improved to 20/100, and at that time yellowish plaques in some of the retinal arterioles were noted for the first time. Their number and intensity increased during the following two weeks, at the end of which period a fundus photograph was made.

At the end of six months the vision had improved to 20/30, the anterior uveitis had subsided, and the chorioretinitic focus had become well defined and markedly pigmented. The arteriolar plaques were still present in large numbers but had lost some of their density and had become less obvious.

CASE 4

S. W., a Negress, aged 27 years, was seen at the Research and Education Hospitals of the University of Illinois with the complaint of blurring left vision for several weeks.

At the time of the first visit the left vision was 20/200 and the left intraocular pressure was 46.0 mm. Hg (Schiøtz). The right eye was normal in every respect. There were numerous left corneal precipitates and aqueous and vitreous were diffusely hazy with a few formed vitreous floaters. A large elevated pigmented chorioretinal lesion occupied the extreme temporal periphery, and almost all retinal arterioles were covered with yellowish plaques at short intervals (fig. 5). The picture was similar to that of Case 13 but the arteriolitis was more widespread.

Physical examination and laboratory tests

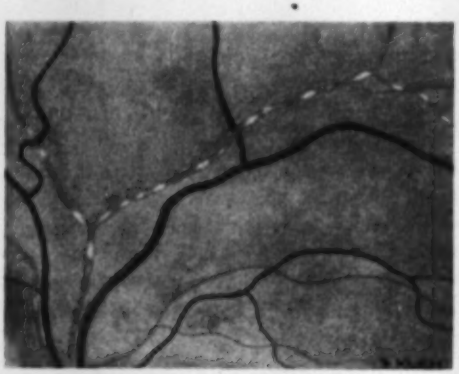

Fig. 5 (Klien). *Case 4.* A Negress, 27 years of age. Yellowish arteriolar plaques at short intervals. Solitary tubercle in extreme periphery of same fundus not shown.

were negative with the exception of a two plus reaction to second strength of PPD (2.0 cm. of marked induration and 4.5 cm. slight induration around it.)

Treatment consisted of streptomycin (0.5 gm. twice a week) and 12 gm. of para-amino-salicylic acid daily.

After 10 months vision of this eye was 20/20 again, the media were clear, and only isolated plaques were left on some of the retinal arterioles. The remaining arterial retinal tree was normal again.

Discussion of Cases 3 and 4

Retinal arteriolitis similar to that of Cases 3 and 4 was first described as a definite clinical picture in 1933 by Kyrieleis.[3] This author thought that the arterial changes were caused by allergic reactions to the toxins of tubercle bacilli. Since then a few reports of cases and some discussions of this condition have appeared in the literature. Muncaster and Allan[4] saw the arteriolitis develop in a young woman as part of a focal reaction from a diagnostic tuberculin injection.

In the most recent discussion of this condition Friemann[5] makes comparisons between arterial and venous involvement of the retinal vessels in uveitis. After reviewing his own cases, and those of Kyrieleis and of several other authors, he comes to the conclusion that one is not dealing with a special type of disease in these vascular involvements but that severity and acuteness of the uveal disease are the deciding factors. The rare participation of the retinal arterioles in the uveal disease points to a more acute process of greater severity than the more common venous involvement, which indicates a chronic and milder course.

We have no knowledge of the histologic picture of the arteriolar plaques, but we do know the histopathologic changes of the venous disease associated with uveitis. Friemann speculates that the lesions in the arterial wall may be predominantly a colloidal reaction with less cellular infiltration than characterizes the venous disease, causing an opaqueness of the vessel wall by changing its index refraction. The fact that the plaques on the arterioles stay in place, never extend beyond the limits of the vessel wall, and gradually fade away without leaving a trace is suggestive of such a process.

The venous lesions, in contradistinction to the arterial changes, are not merely spots in the vessel wall but actually nodular thickenings of the wall, parts of which may even become detached from the affected vein and may then lie free in the posterior vitreous (Friemann,[5] Van der Heuvel[6]).

III. Periphlebitis and Phlebitis

A typical example of participation of the retinal veins in uveal disease is the neuroretinitis papulosa (Fuchs'[7]), in which periphlebitis is part of the characteristic triad of findings.

Case 5

S. M., a man 37 years of age, entered Billings Hospital complaining of failing right vision for the past two years. Two years ago the right eye was painful and red for several months with markedly reduced vision. The inflammation gradually disappeared but the right vision remained defective.

There was a history of syphilitic infection at the age of 16 years and of a reinfection at the age of 30 years.

Wassermann and Kahn tests were four plus at the time of the first examination.

The right fundus showed an old disseminated chorioretinitis, a postneuritic optic atrophy, and evidence of old and of active periphlebitis or phlebitis (figs. 6 and 7). The number of hemorrhages suggested that at least in some places the entire venous wall was diseased.

Case 6

A woman, 48 years of age, had a tuberculous uveitis of her right eye of several years' duration. She developed a secondary glaucoma for which an iridectomy

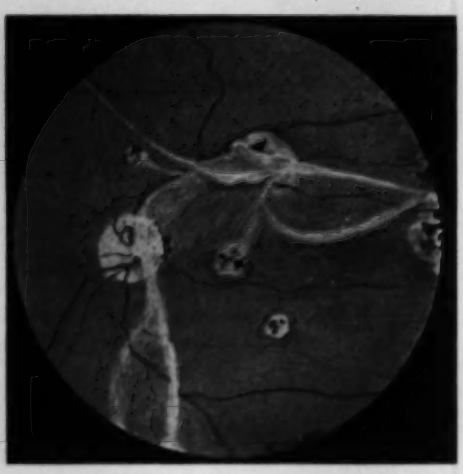

Fig. 6 (Klien). *Case 5.* Neuroretinitis papulosa. A man, 37 years of age. Old disseminated chorioretinitis, postneuritic optic atrophy, and retinitis proliferans.

Fig. 7 (Klien). *Case 5.* Still active retinal phlebitis in same fundus as shown in Figure 6.

was eventually performed. The eye did not become quiescent and, since the vision was only light perception, it was removed.

The histopathologic examination revealed a localized scleritis and episcleritis on one side. There was diffuse infiltration with lymphocytes and plasma cells of iris and ciliary body. The iris was quite atrophic. Its anterior surface and the posterior surface of the cornea were lined with numerous precipitates and individual round cells. There were extensive anterior and posterior synechias. The lens equator was injured at the site of the postoperative scar. Numerous precipitates consisting of lymphocytes and monocytes were suspended in the aqueous of the posterior chamber, lining some of the zonular fibers, and in the vitreous. There was an extensive vitreous detachment.

Foci of specific granulation tissue were found only in the choroid (fig. 8). Some were small, leaving the pigment epithelium intact; others occupied the entire thickness of the choroid, causing extensive chorioretinal adhesions.

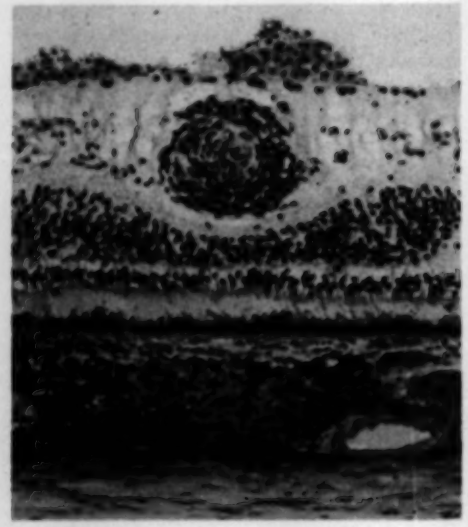

Fig. 8 (Klien). *Case 6.* A woman, 48 years of age, tuberculous uveitis. Focus of specific granulation tissue in choroid, inner surface of retina lined with pseudoprecipitates and fibrillary substance, perivenous infiltration with lymphocytes in retina.

The inner surface of the retina was lined by rows of preretinal precipitates which were held together by a stringy fibrillary substance (fibrin?). Many of the retinal veins had walls densely infiltrated with lymphocytes, plasma cells, and plasma-cytoid cells, and a markedly narrowed lumen, deserving of the term of phlebitis rather than periphlebitis.

IV. Capillaritis

In some cases of participation of the retina in uveal inflammation cellular infiltration is present not only in and around the retinal veins but also along the capillaries, producing a most striking picture.

The superficial capillaries and the connecting branches to the deeper ones become extremely well visible, and in the macular region also the third capillary layer, as well as the third and fourth peripapillary capillary bed, become discernible. The tissue around these capillaries becomes edematous. The edema is particularly pronounced in the nerve fiber layer which becomes markedly thickened (fig. 9).

Case 7

E. L., a boy, aged 14 years, entered the Cook County Hospital with a penetrating injury of his right eye, sustained when a rock, thrown against the eye, broke his glasses.

There were two corneoscleral lacerations, with prolapse of iris, which were repaired by excision of the prolapsed iris and direct suturing. The anterior chamber reformed promptly, but the lens became cataractous and a severe anterior uveitis developed.

One month after the injury the fellow eye became mildly injected and cells appeared in its aqueous. The inflammation receded under systemic cortisone and ACTH therapy. In the meantime a hyphema developed in the right eye filling the entire anterior chamber. During its slow absorption the light projection in this eye became faulty and the globe was removed.

Histologically the diagnosis of sympathetic ophthalmia was confirmed. In addition to the granulomatous involvement of the entire uvea in locations typical of sympathetic ophthalmia, there was a wreath of giant cells mixed with polymorphs around and penetrating into the remnants of the lens. Inside of the curled-up lens capsule there was a circumscribed abscess consisting of necrotic material and polymorphs, a picture typical of a lens induced inflammation.

The most striking lesion was found in the retina, where all the capillary layers had become extremely distinct due to infiltration

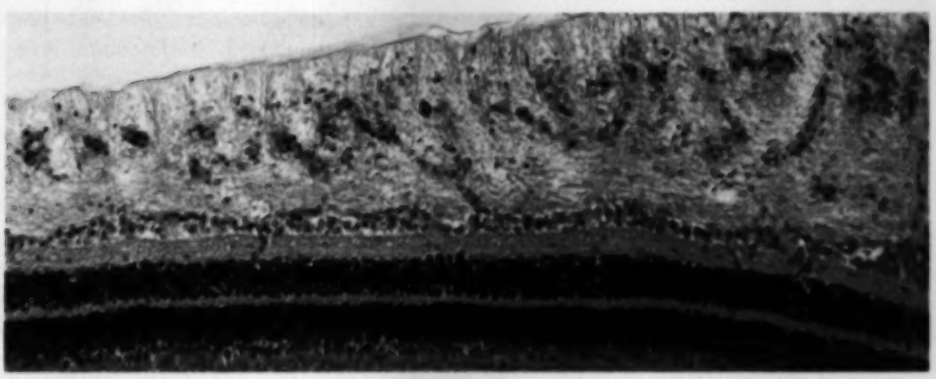

Fig. 9 (Klien). *Case 7.* A boy, 14 years of age, sympathetic ophthalmia after penetrating injury, superimposed lens-induced inflammation. Inner layers of retina edematous, lymphocytic infiltration along all capillaries. Capillaritis probably part of lens-induced inflammation.

with lymphocytes along their course (fig. 9). The inner layers of the retina were edematous, a picture very similar to that of serous retinitis. These retinal changes were thought to be part of the lens induced inflammation and not due to sympathetic ophthalmia.

Combination of two distinct inflammatory processes of a different type as in this eye is rare but not unknown. Lens induced inflammation has been found superimposed upon tuberculous uveitis and sympathetic ophthalmia (Scheffler,[8] Klien,[9] and others). The co-existence of two conditions in the pathogenesis of which allergy or hypersensitivity plays a role is really not surprising.

It is doubtful whether the combination of sympathetic ophthalmia and lens induced inflammation can be diagnosed clinically since the therapeutic response of these two conditions is probably similar, especially if the lens induced condition is a sensitivity reaction and not merely of toxic origin.

The resemblance of the retinal capillaritis and periphlebitis and of the associated edema of the inner retina layers, in this case to the histopathologic picture of retinitis serosa as shown in Case 2, suggests that they might be only different degrees of the same process, and that, at least in the posttraumatic or postoperative cases, retinitis serosa might be part of a low grade, lens induced inflammation.

V. Central angiospastic retinopathy

The lesions of the retinal vessels associated with uveal disease demonstrated in the foregoing were organic in nature and not of sudden onset. In some cases of uveal disease sudden visual disturbances point to a functional disorder of the retinal vessels which may produce the subjective and objective picture of central angiospastic retinopathy, indistinguishable from the idiopathic type.

The importance of this complication lies in the possibility of incorrect diagnosis and treatment should the uveal disease be atypical or inconspicuous and the clinical picture be dominated by the associated retinal condition.

Case 8

D. E., a dental student, 26 years of age, came to the Research and Educational Hospitals of the University of Illinois complaining of metamorphopsia and slight diminution of the left vision of a few days duration. The right vision had been poor for the past three years. Failure of the right vision had started in a manner similar to the recent left visual disturbance. At that time the patient was seen in another city where the diagnosis of juvenile disciform macular degeneration of the right eye was made.

At the time of the first visit the right vision was 20/200, the left vision 20/20+3. The left visual disturbance consisted, in addition to the metamorphopsia, of a relative positive central scotoma in the form of a yellowish discoloration around the fixation area.

The anterior segments were normal in both eyes, the media clear.

In the right fundus there was in the center of the macula an elevated fibrotic mass about 0.75 disc diameters in size. Aside from a few small, disseminated, old, atrophic chorioretinitic scars, the remaining fundus was normal.

The left fundus revealed also a few similar, old, disseminated chorioretinitic areas. Adjoining the upper nasal margin of the left disc a white fibrotic lesion, about 0.5 disc diameters in size, projected slightly above the retinal level. In the macula there was a faintly but definitely outlined cystic appearing elevation with fine white dots dispersed over its anterior surface, presenting the typical picture of an acute central angiospastic retinopathy.

One week afterward the left vision was still 20/20+3. At that time, only the inferior outline of the macular cyst was still sharp and most of the white dots had disappeared. A circumscribed, slightly elevated gray area,

one-third disc diameter in size, with a narrow hemorrhagic fringe was then noted in the upper macular area.

Within the following 10 days the left vision diminished to 20/200 due to seepage of hemorrhage from this lesion into the fovea centralis.

Physical examination revealed an allergic rhinitis but was otherwise negative. Laboratory tests were negative with the exception of an inconclusive reaction to histoplasmin (a 1.5 cm. area of erythema).

The acute lesion gradually underwent a transformation from an ill-defined grayish to a sharply outlined yellowish white area, which off and on shows a fringe of deep hemorrhage.

Throughout the two years of observation the patient has been a therapeutic problem with vision in this eye now varying from 20/40 to 20/100 with recurring seepage of hemorrhage into the fovea.

The diagnostic difficulties in this case were increased by the fact that the initial visual disturbance was of retinal origin, namely, metamorphopsia with only a slight decrease in vision, and a relative positive scotoma, with a fundus picture of a central serous retinopathy. It is possible that the first sign of the choroidal focus was also present at the time, but the acute clinical picture was so overwhelmingly and typically that of a retinal disease that it did not register with the observers.

Similar findings in the other eye three years prior to the onset of the left visual disturbances may have been the basis for the diagnosis of juvenile disciform macular degeneration made at the time.

The disseminated old chorioretinitic lesions associated with the fresh focus above the left fovea left little doubt in our minds that we were dealing with an inflammatory process, of undetermined origin, with a protracted course over many years, coming to the patient's attention only through incidental involvement of the macular areas.

The presence in his fundi of atrophic and hypertrophic chorioretinitic scars suggests that some of the foci at the time of their activity were also associated with hemorrhages, either repeated ones as in the present lesion or at least one massive hemorrhage. The expectation regarding the end-result of the present focus would be for a hypertrophic scar similar to those at the left disc and the right macula.

CASE 9

K. J., a school teacher, aged 61 years, gave the history of sudden marked impairment of her right vision one year prior to her first visit at Billings Memorial Hospital. This first attack lasted only a few minutes but several days afterward she noted that objects appeared to the right eye as if seen through a veil.

She was told by her physician that there was some disease of the retinal arteries in this eye and was kept on rutin medication for the following four to five months. During the latter part of this time photographs of the right fundus were made (fig. 10). These pictures show in addition to old lesions on the nasal side of the optic disc, one subacute and one acute focus in the macular region. The subacute lesion nasally and above to the foveal region, without edema but

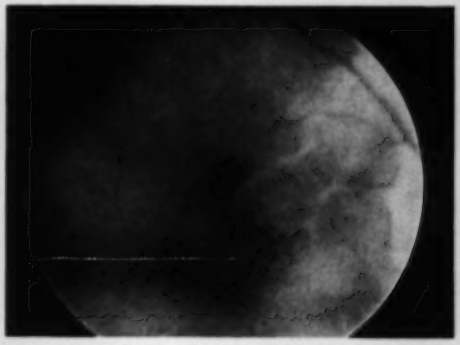

Fig. 10 (Klien). *Case 9.* Chronic active inner chorioiditis. A woman, 61 years of age, right fundus five months after initial visual disturbance. Old lesion: broad grayish band with yellow, well-defined borders. At its inferior end the subacute lesion: still faintly circular with faintly pigmented rim. Acute lesion: circular, gray, ill-defined.

still showing its circular shape, had begun to develop a faint pigment rim. The acute focus, inferior nasally to the subacute lesion and also circular in shape, was ill defined, and grayish due to overlying edema. The subacute lesion was situated at the end of a broad, flat, grayish band with yellowish, well-defined borders, which may have arisen from confluence of similar lesions. The optic disc and retinal vessels appeared normal.

Since the beginning of the visual disturbance up to the present time the patient had a sensation of "sparkles" before the right eye, and after six months she had noted for the first time a defect in the visual field just to the right of the point of fixation. There was never pain or redness of this eye.

At the time of her first visit the right vision was 3/200, the left vision 20/15. The left fundus was normal. In the right fundus the optic disc and retinal vessels appeared normal. Around the disc, either directly adjacent to it or a short distance from it, there was a yellowish lobulated, apparently atrophic zone. From it arose fingerlike extentions, which again branched out into different directions. Not all of these areas showed the same degree of atrophy. In some portions only the large choroidal vessels were left, the inner half of the choroid and the pigment epithelium being completely destroyed. Other portions, especially at the end of the fingerlike extensions, were only slightly lighter than the normal fundus and covered with fine dusty pigmentation.

The macular area was occupied by a similar lesion, which altogether appeared less atrophic than the nasal lesions, but which also branched out into the periphery. Throughout it there was a faint pattern of a choroidal vascular network visible, as if only pigment epithelium and perhaps the choriocapillaris were missing (fig. 11).

At the ending of one of the superior temporal branches there was an ill-defined, 0.5 disc diameter in size, round yellowish gray area, resembling the acute focus in the earlier fundus photograph which was considered to be an acute chorioiditic focus. A similar smaller lesion was situated in the inferior temporal perimacular region.

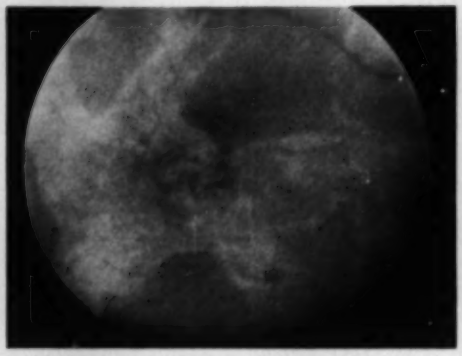

Fig. 11 (Klien). *Case 9.* Right fundus one year after initial visual disturbance, Vision: R.E., 3/200. Macular lesion with fingerlike extensions similar to broad band in first photograph.

The macular retina exhibited coarse pigment migration into it and considerable cystic degeneration.

A diagnosis of chronic active inner choroiditis was made.

General medical examination and laboratory tests were negative with the exception of a 3.0 cm. induration and erythema following 1:10,000 old tuberculin intracutaneous test.

Therapy with isoniacid (300 mg.) and para-amino-salicylic acid (10 gm.) daily was started.

During the past two months no fresh lesions have developed and the two previously observed active lesions have become sharply defined and covered with a fine dusty pigmentation.

The vision is still 3/200, the sensation of "sparkles" is still present, but less intense and frequent. A peculiar subjective feature is the presence of positive scotomas, which the patient is able to draw, which do not exactly correspond to the visible lesions but have their principal shape (fig. 12). This suggests that within some parts of the more recent lesions there is an incomplete destruction of the percipient retinal elements,

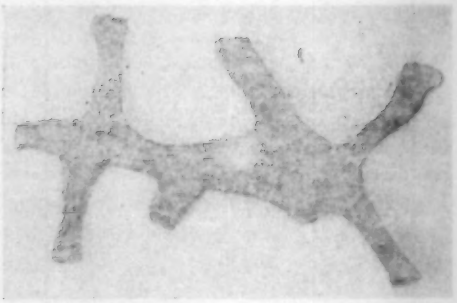

Fig. 12 (Klien). *Case 9.* Positive scotoma drawn by patient, of general shape as fundus lesions.

and some sort of retinal participation in the choroiditic process leading to opacities in front of the rods and cones.

The diagnostic problem in this case was made more difficult by the fact that the initial visual disturbances pointed to a retinal vascular disease.

Since the patient was not examined by me at this stage, I may merely speculate upon the nature of this vascular process. It could have been a spasm of the larger arterioles or of the perimacular arterioles and precapillaries such as occurred in Case 8. It must have been functional because no organic lesions of the retinal arterial tree or of the optic nerve are visible at this time.

The important aspect of this case to be emphasized is the fact that retinal vascular phenomena associated with the choroiditis led to an incorrect diagnosis and deprived the patient for six months or more of therapy for the basic condition.

The frequency of association of functional retinal vascular disturbances with uveal disease is not known. It may be quite common, if one looks for it. A further illustration of it is the following case, which was not a diagnostic problem.

Case 10

G. N., a man, aged 37 years, noted blurring of his right vision for the first time seven months ago. The left vision had always been poor so that any diminution of the right vision was noted immediately. An acute choroiditis was then diagnosed. After receiving a series of streptomycin injections the vision cleared.

Blurring of the right vision was again noted a week prior to his first visit at Billings Memorial Hospital. At that time, the right vision was 20/50 and no Jaeger, the vision of the left amblyopic eye was also 20/50. The left eye was otherwise normal.

The right globe was pale, but with the slitlamp many cells were visible on the posterior corneal surface and in the aqueous. There was a slight vitreous haze but the fundus was well visible. Optic disc and retinal vessels were normal. The macular

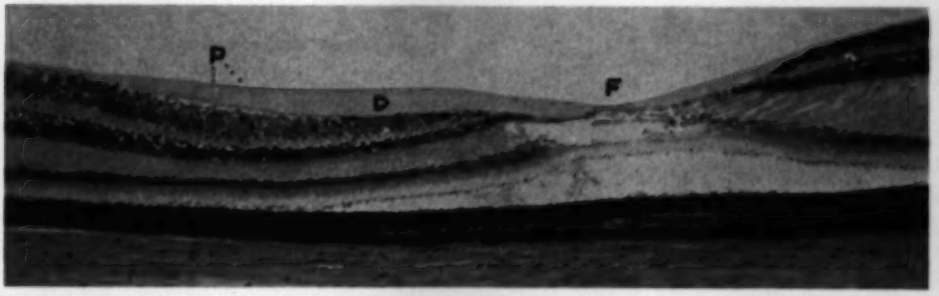

Fig. 13 (Klien). *Case 9.* A man, 41 years of age. Right eye: malignant melanoma of the iris and ciliary body, central angiospastic retinopathy. Right vision 20/50 at time of enucleation. General view of macula: extensive detachment of internal limiting membrane (D), region of fovea centralis (F), small tags of cellular debris (P) on outside of internal limiting membrane. Circumscribed destruction of neuroepithelium and corresponding second and third neuron in foveal and perifoveal region.

area was edematous and there was a grayish deposit in and temporal to the fovea centralis with a small hemorrhage, which was apparently on the temporal slope of the clivus. The first impression, looking just at the macular area, was that of a retinal disease. Examination of the remaining fundus, however, revealed a fresh solitary tubercle 2.5 disc diameters temporal to and slightly below the macular area, 1.5 disc diameters in size, and an old atrophic lesion of the same size just above it. The retinal edema around the fresh tubercle was not connected with the macular edema.

The patient's reaction to 1:10,000 strength of PPD was four plus and therapy with isoniacid and para-amino-salicylic acid was started promptly.

Within 10 days the macular hemorrhage had absorbed and the gray deposit was confined to the bottom of the fovea centralis. Within another 10 days the fovea and macula had regained an entirely normal appearance with a vision of 20/20 and J1.

The interesting feature of this case is the initial diminution of vision out of proportion to the faint haze of the media due to the uveal process. Specially questioned about it, the patient recalled that the vision had become poor quite suddenly.

The macular lesion had no direct connection with the solitary tubercle, but resembled an angiospastic retinopathy with hemorrhagic extravasation.

The improvement of the vision was also rapid and out of proportion to the slow improvement of the uveal disease. Most of the visual impairment appeared to have been due to a functional vascular disturbance associated with the uveal disease. The latter was, however, so obvious that no error in the basic diagnosis was possible.

The next, histologically studied case illustrates an apparently functional disturbance of some of the intraocular vessels, including those of the retina, associated with a malignant melanoma of the iris.

CASE 11*

R. E., a man, 46 years of age, stated that three years prior to the first examination by his ophthalmologist he suffered a contusion of the left eye for which he was under a physician's care for about a month. Soon after this injury he noted a brown spot in the nasal half of the left iris. The vision of this eye was not affected by it and remained normal for two years. For the past year only, there occurred at infrequent intervals a slight blurring of the left vision. These attacks lasted a few days, but vision always returned to normal. For the past month, however, there were times when the left vision was completely blurred for a few hours, remaining impaired in the intervals.

The corrected vision of the completely normal right eye was 20/20, corrected left vision was 20/50. The left globe was pale. The temporal half of the iris was blue, the nasal half was transformed into a brown tumor mass.

The left fundus was well visible and appeared normal. The left intraocular pressure was 35 mm. Hg (Schiøtz). The globe was removed with the diagnosis of malignant melanoma of the iris.

The specimen was fixed in formalin and was cut in the horizontal plane. Lens, ciliary body, retina, and choroid were in situ. On the nasal side a yellowish brown tumor filled the space between cornea, lens, and corona ciliaris.

The tumor was composed of spindle cells (subtype B) and epithelioid cells, and was pigmented only in scattered areas. It had completely infiltrated the nasal iris and broken through the posterior surface of the iris near its root. It also had begun to infiltrate the anterior portion of the ciliary body. From the free surface of the tumor small groups of cells had separated and become implanted on the inner surface of the

* I am indebted to Dr. Hershel S. Smith, Bloomington, Indiana, for the pathologic specimen and the excellent history.

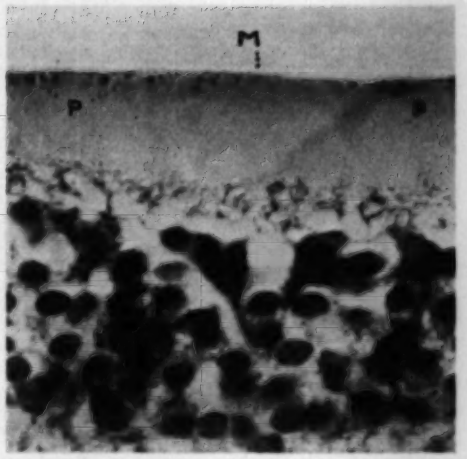

Fig. 14 (Klien). *Case 11.* High-power view of deposits (P) on outside of internal limiting membrane (M), possibly the cause of white punctate lesions, often visible clinically in central angiospastic retinopathy.

ciliary epithelium and in the opposite chamber angle where they had started to infiltrate the root of the iris and the anterior surface of the ciliary body.

On the free surface of the tumor there were small hemorrhages. Isolated groups of erythrocytes were also visible in the anterior chamber, posterior chamber, and anterior vitreous. The temporal iris had normal structure but contained scattered small stromal hemorrhages. There were no signs of inflammation in iris, ciliary body, or choroid. The optic nerve was normal excepting a slight retrodisplacement of the cribriform plate.

The most striking alterations of the posterior segment were in the macular retina. The retina elsewhere was normal. In the fovea centralis and its immediate neighborhood the neuroepithelium was destroyed. There was an extensive detachment of the internal limiting membrane by a homogeneous (serous) coagulum. Attached to the outside of the membrane there were a few small, inconspicuous granular deposits, either cellular debris, or tags of tissue from the inner surface of the nerve-fiber layer, or coagulated albuminous or fibrinous material present in the transudate. There was edematous imbibition of all retinal layers, especially pronounced in Henle's fiber layer, but no thickening of the nerve fiber layer. From Henle's layer the transudate had reached and disorganized the cone nuclei and the neuroepithelium, and a small amount of it had seeped into the subretinal space (figs. 13 and 14). A small conglomeration of free erythrocytes was lying on the nasal incline of the clivus. Signs of inflammation in retina or choroid were absent. There was no irritative proliferation of the pigment epithelium as yet.

These findings were those of a functional vascular disturbance in the macula as previously described (Klien[10]). The destruction of the foveal cones indicated repeated attacks or one severe, prolonged one. The history of the visual disturbance given by the patient suggests the former.

Also in this case the central angiospastic retinopathy was probably not an independent phenomenon, but a secondary manifestation caused by toxic properties of the growing uveal tumor, which were indicated also by the multiple stromal hemorrhages in the iris.

A case similar to the one just described came to my attention during the meeting of the Ocular Pathology Club in April, 1956. Dr. W. E. Fry reported the clinical and histopathologic findings in the eye of a woman, aged 35 years, which was removed because of a malignant melanoma of the choroid located in the temporal equatorial region.

The initial complaint which brought the patient to the ophthalmologist was a slight blurring of the central vision in this eye. A paracentral scotoma was found and the macular region presented the typical picture of a central angiospastic retinopathy.

While the histopathologic findings in the enucleated eye of Dr. Fry's patient were no longer typical of a central angiospastic retinopathy or choriopathy because of the

extensive retinal detachment, there seems to be little doubt that the patient had for some time prior to the enucleation the clinical symptoms of a central angiospastic retinopathy.

Possibly, this association of a functional vascular disturbance with malignant uveal melanomas of varying degrees of toxicity is more common than the lack of such observations in the literature permits us to believe. Dr. Fry's advice to examine carefully the fundus periphery of eyes with central angiospastic retinopathy or chorioretinopathy for hidden incipient malignant melanomas is seconded by me.

Summary and Conclusions

Retinal involvements associated with uveal disease fall into several categories, namely, inflammatory, functional, and degenerative. Representative cases, belonging to the first two, were demonstrated in the foregoing. The obvious direct extension of an inflammatory choroidal focus into the adjacent retina was disregarded, as only lesions posing a diagnostic problem were intended for discussion.

The inflammatory manifestations under consideration may be subdivided into:

1. Those, encountered predominantly in the retinal stroma or on the retinal surface as in retinitis serosa, and

2. Those, affecting mainly the retinal vessels in the form of arteriolitis, phlebitis, or periphlebitis, and capillaritis.

The diagnostic importance of retinitis serosa, which is a relatively rare affliction, lies in its after effects upon vitreous and retina. Therefore, an attempt was made to circumscribe more definitely its clinical picture. It occurs together with a postoperative or posttraumatic nonspecific infiltrating iritis or with an endogenous iridocyclitis of similar type and long duration. There is some evidence that in all these instances it may be part of a low grade lens induced inflammation, in which steroid therapy may be useful and may prevent some of the late complications.

Among the organic vascular lesions of the retina associated with uveitis, arteriolitis seems to be the diagnostically most helpful, having so far been found only in frankly tuberculous disease.

Phlebitis and periphlebitis may accompany a variety of uveal lesions. They are an essential part of the triad of findings in neuroretinitis papulosa of syphilitic origin, and they are not uncommon in tuberculous uveitis and in lens induced inflammation.

Retinal capillaritis is part of the picture of lens induced inflammation when it extends to the posterior segment. It may also appear in retinitis serosa.

The functional disturbances of the retinal vessels associated with uveitis confuse rather than aid the diagnosis. The picture of central angiospastic retinopathy with serous or hemorrhagic extravasations may overshadow the symptoms of the uveal disease, especially in the initial stages, with the inherent danger of an erroneous diagnosis. Thus, the functional vascular disturbances, allergic or toxic in origin, are the most important retinal participation in uveal disease to remember when interpreting the subjective and objective clinical picture.

The degenerative lesions of the retina associated with uveal disease and their consequences of retinoschisis, cyst formation, inner and outer cyst ruptures, and retinal separation are discussed in Part II of this publication.

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HISTOPATHOLOGY OF KERATOCONUS*

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New York

Keratoconus or conical cornea has been known to ophthalmologists for many years. Its etiology is, however, still obscure. Although the literature on keratoconus is voluminous, reports on the histopathology of this disease are few. Since keratoconus in itself rarely leads to enucleation, paucity of pathologic material, even of well-developed keratoconus, is not surprising.

Bowman,[1] in 1859, was the first to report the condition pathologically. He removed an eye with keratoconus which was examined by Hulke. Hulke found the central part much thinner than the periphery. There was gradual thinning toward the apex. The normal lamellae of the stroma were replaced by a web of caudate and nuclear fibers. Bowman's membrane was intact, but thinned and wrinkled over the apex. Descemet's membrane was unchanged.

Brailey,[2] in 1876, reporting on the trephined apex of a cornea from a case of keratoconus, found Bowman's membrane intact, and spaces in the superficial layers of the epithelium.

Salzmann,[3] in 1908, presented a careful histologic study of an eye affected by keratoconus. The examination showed the following findings: (1) A thinning of Bowman's membrane with many gaps in its continuity, (2) the gaps filled with an "elastoid" connective tissue similar to that found in pinguecula, (3) at the apex of the cone the deep layers of the stroma revealed some tendency to split, with the formation of lacunae longer than those seen in normal stroma, (4) a considerable gap in Descemet's membrane at the region of the greatest ectasia, and (5) the endothelium at the apex appeared normal except at the edges of the ruptured Descemet's membrane where the cells appeared larger.

Vogt's[4,5] histologic examination of the apex of two eyes with keratoconus in 1937 and 1939 showed, within the thinnest portion, a waving of the stroma representing the deep striae in keratoconus. In the first case this was present only in the posterior stroma but in the later case this condition was present through the entire thickness.

Terry and Chisholm,[6] in 1940, were apparently the first authors in this country to report a histopathologic examination in four cases with keratoconus. In only two of these cases were the changes of the type described by Salzmann present. As the other two cases were complicated by adherent leukoma, staphyloma, and secondary glaucoma, no findings specifically characteristic of keratoconus as reported by Salzmann were noted.

After 1940, the histopathology of keratoconus received very little attention in the

* From the laboratory of The Eye-Bank for Sight Restoration, Inc., Manhattan Eye, Ear, and Throat Hospital; aided by Grant B 153 from the National Institutes of Health and by the Josiah Macy, Jr., Foundation.

literature, until 1954, when Hervouët[7] made a detailed description of histopathologic findings in keratoconus, accompanied by many beautiful photomicrographs. The material was obtained during corneal transplantation from nine patients with keratoconus. The author especially stated that histopathologic examination of early stages of keratoconus was badly needed. He pointed out that numerous dehiscences of Bowman's membrane on top of the undulation and the extraordinary invagination of Descemet's membrane were two pathognomonic signs to give a precise pathologic diagnosis to keratoconus.

Evidently in the past all pathologic material studied represented late stages of the disease after complications. Since corneal transplantation in cases of keratoconus presents such a favorable prognosis, particularly if operated in the relatively early stages, there should be an increasing source of material for histopathologic study to understand the mechanism of the development of the disease.

Material and Methods

Our material consisted of pathologic discs removed at keratoplasty. A total of 65 such discs collected from the Manhattan Eye, Ear, and Throat Hospital* were studied. Each disc was fixed immediately in 10-percent neutral formol-saline solution, and then embedded in paraffin. The fixed tissue was bisected and sectioned serially in both the transverse and surface planes. In some instances the posterior portion of the disc involving the endothelium, Descemet's membrane, and as little as possible of the stroma was mounted as a flat preparation to study the endothelium. Serial sections were stained with hematoxylin and eosin, Verhoeff's elastic tissue method, toluidine blue, van Gieson, and periodic acid-Schiff stain, and were prepared for both quantitative and qualitative correlation of the pathologic process found.

* We are indebted to Dr. R. Townley Paton for supplying most of the material that made this study possible. We wish to acknowledge the technical assistance of Mrs. J. Solomon.

Histologic findings

Epithelium

The changes in the epithelium were various, and all layers may be involved; however, the superficial layers are frequently normal, especially at the base of the cone. In the deep layers of the epithelium, the basal cells are usually affected at a relatively early stage. In places they appear pale, edematous, and swollen with pyknotic nuclei (figs. 8 and 11). Sometimes they lose their cylindric shape and palisading arrangement (fig. 11). In more advanced stages the basal cells eventually disappear, the epithelium being represented by only one or two layers of superficial flattened cells (fig. 12), lying on an altered basement membrane, Bowman's membrane, or directly on the anterior stroma.

Basement membrane

With periodic acid-Schiff stain, multiple dehiscences in the basement membrane are noted in the sites corresponding to the lesions found in Bowman's membrane or in the epithelium (figs. 4, 9, 10, 11, and 14). In early lesions the basement membrane shows fragmentation in the areas of degenerated Bowman's membrane without discernible change in its overlying epithelium (figs. 2 and 3).

Bowman's membrane

The most interesting feature encountered in this study is the lesion in Bowman's membrane which varies according to the different stages of the disease. Swelling and fibrillation of this membrane were noted (figs. 1, 2, and 3), which we have not seen reported in the literature. In these lesions, the fibrillar degeneration of Bowman's membrane appeared as extremely fine fibrils instead of having a normal homogeneous appearance (figs. 2 and 3). The fibrillation is

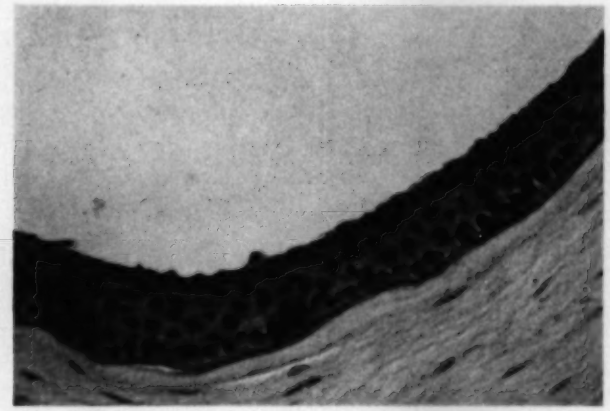

Fig. 1 (Chi, Katzin, and Teng). Multiple lesions in the basement membrane of the epithelium and in Bowman's membrane. Early changes: (1) Fragmentation of the basement membrane. (2) Swelling and fibrillar degeneration of Bowman's membrane. (×112, BP 79-1, periodic acid-Schiff stain.)

better shown with phase-contrast microscopy than under ordinary light microscopic examination.

In some instances the diseased areas of Bowman's membrane show increased staining with toluidine blue, as well as an accumulation of metachromasia (fig. 7). These areas also show increased intensity of staining with periodic acid-Schiff (figs. 6, 8, 9, 10, and 11). These changes appear to indicate the first lesions in Bowman's membrane. As the process of fibrillation continues, the fibrils become coarse and extend to the adjacent stroma (fig. 5).

In late stages Bowman's membrane is gradually destroyed and keratoblasts migrate to these sites, followed by newly formed connective tissue (figs. 12, 13, and 15). This

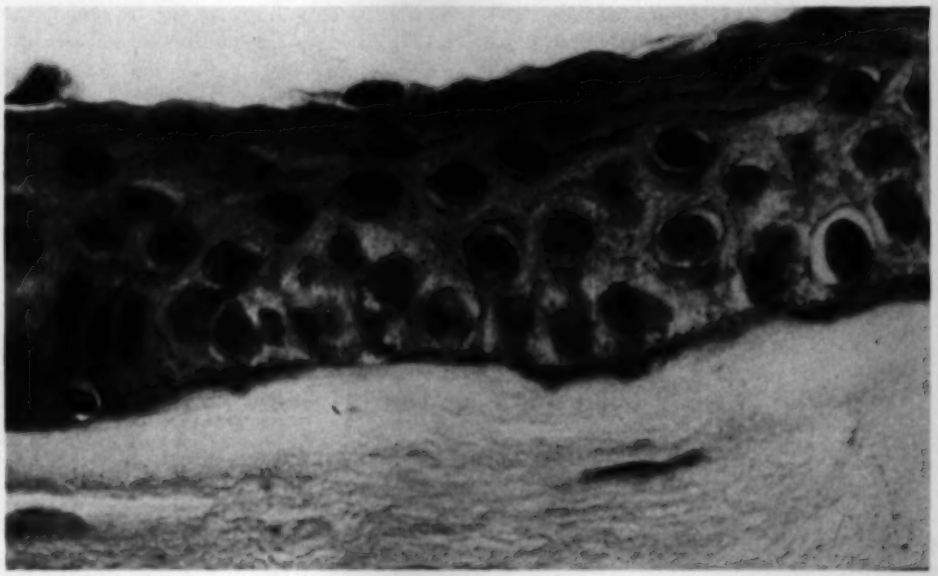

Fig. 2 (Chi, Katzin, and Teng). Higher magnification of the central lesion of Figure 1, showing fragmentation of the basement membrane and swelling and fibrillation of Bowman's membrane. (×500, BP 79-1, periodic acid-Schiff stain.)

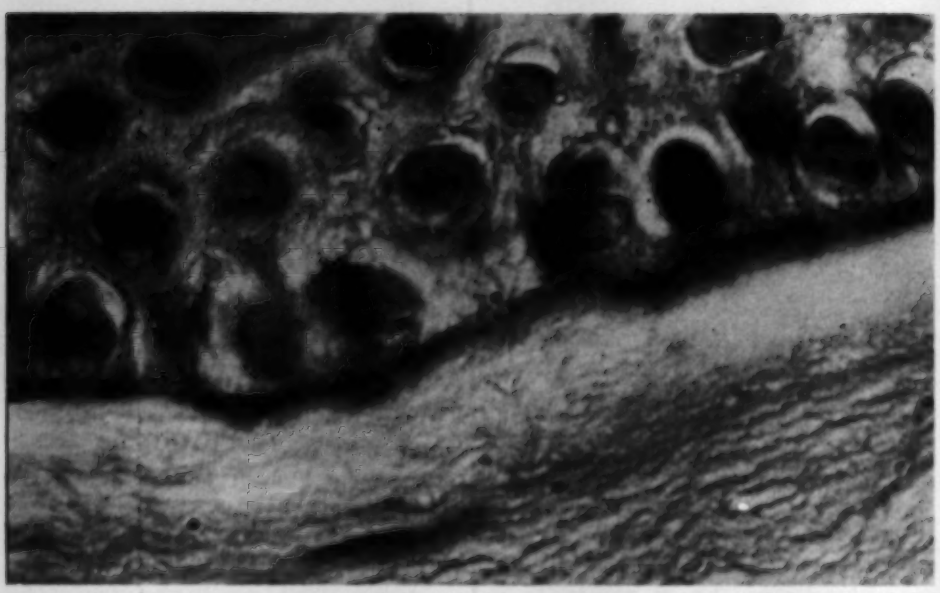

Fig. 3 (Chi, Katzin, and Teng). Higher magnification of Figure 2. (×800.)

results in the characteristic lesion of Bowman's membrane which shows multiple narrow gaps and a wavy appearance (fig. 15).

The gaps in Bowman's membrane are filled either with a newly formed connective tissue consisting of a focal accumulation of keratoblasts or with epithelium (figs. 13 and 14). The gaps filled with the connective tissue are nearly always on the crest of the wave and the downgrowths of the epithelium into the gap are in the trough of the wave. In study of the serial sections the gaps of Bowman's membrane appear as lines and holes which vary in length and size. The gaps are not limited to the apex of the cone; they are not infrequently found

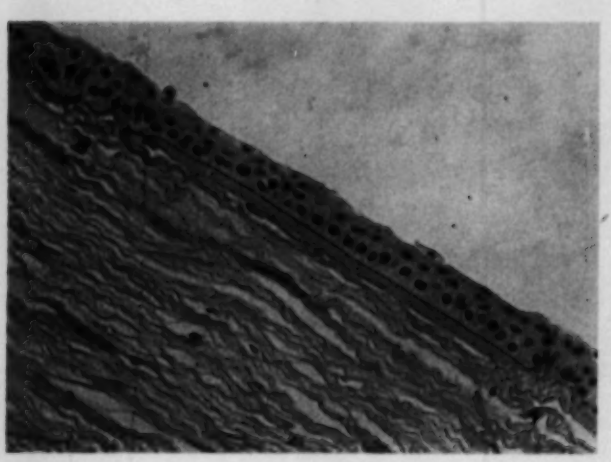

Fig. 4 (Chi, Katzin, and Teng). Two areas of degeneration in the basement membrane and Bowman's membrane. (A) Complete absence of basement membrane; the fibrillar change in Bowman's membrane has become coarse and extends to the adjacent stroma. (B) Gap in the basement membrane and Bowman's membrane filled by newformed connective tissue. (×90, BP 32, periodic acid-Schiff stain.)

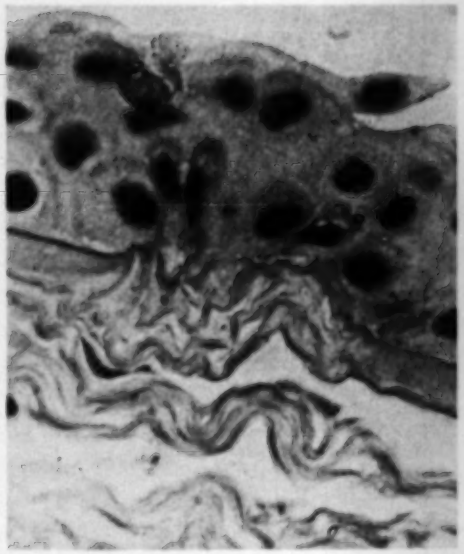

Fig. 5 (Chi, Katzin, and Teng). Higher magnification of area (A) of Figure 4. (×500, BP 32, periodic acid-Schiff stain.)

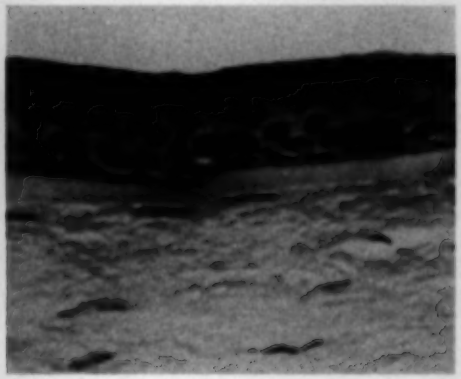

Fig. 6 (Chi, Katzin, and Teng). Early lesion in Bowman's membrane shows changes in staining characteristics—increased intensity with periodic acid-Schiff stain. (×160, BP 112-3, periodic acid-Schiff stain.)

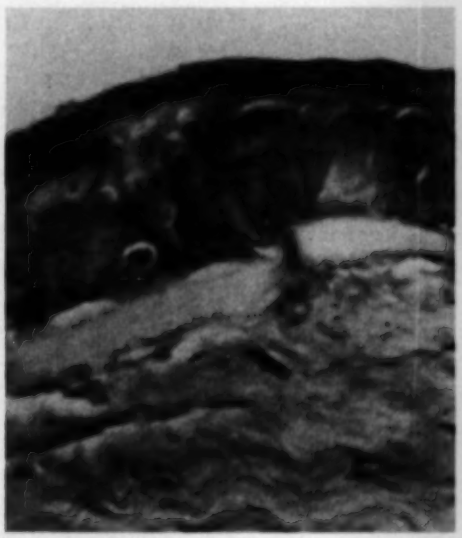

Fig. 7 (Chi, Katzin, and Teng). Area shows change in the staining character of Bowman's membrane with accumulation of metachromatic substance. (×180, BP 98-1, toluidine blue stain.)

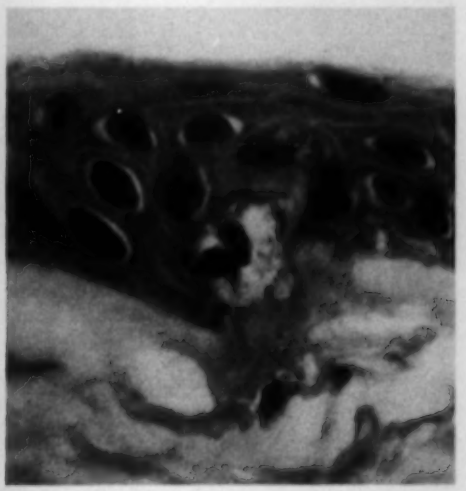

Fig. 8 (Chi, Katzin, and Teng). (1) Epithelium reduced to three layers. Degenerated cells appear pale with pyknotic nuclei. (2) Increased staining intensity of Bowman's membrane with periodic-acid-Schiff stain. (×500, BP 12-27, periodic acid-Schiff stain.)

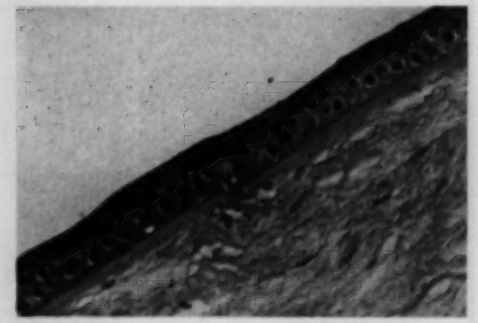

Fig. 9 (Chi, Katzin, and Teng). Focus of destruction in the basement membrane and increased intensity with periodic acid-Schiff stain of Bowman's membrane; migration of keratoblasts to the site. (×210, BP 100-23, periodic acid-Schiff stain.)

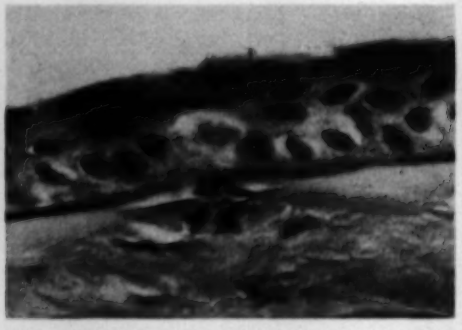

Fig. 11 (Chi, Katzin, and Teng). (1) Epithelium reduced to two to three layers and edematous in appearance. (2) Bowman's membrane showing increased intensity with periodic acid-Schiff stain; focal concentration of keratoblasts. (×500, BP 112-7, periodic acid-Schiff stain.)

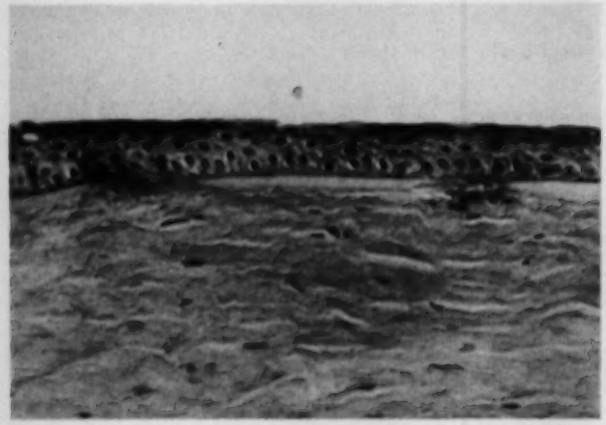

Fig. 10 (Chi, Katzin, and Teng). Two areas with loss of basement membrane; increased intensity with periodic acid-Schiff staining of Bowman's membrane and focal accumulation of keratoblasts. (×160, BP 100-3, periodic acid-Schiff stain.)

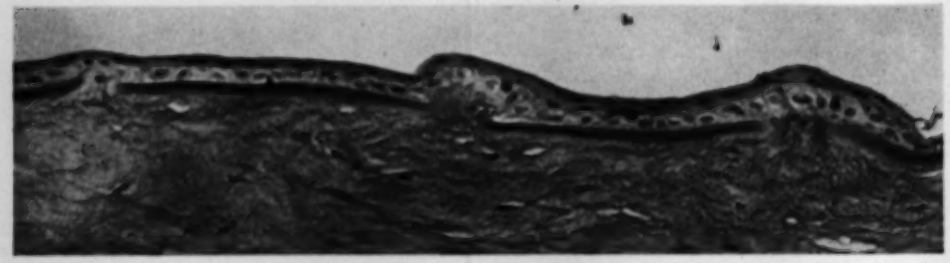

Fig. 12 (Chi, Katzin, and Teng). (1) Multiple gaps in Bowman's membrane with migration of keratoblasts to these sites. (2) Epithelium reduced to two or three layers. (×90, BP 117-6, Van Gieson stain.)

at the base. In more advanced cases the gaps in Bowman's membrane extend more broadly and deeply, involving more stroma. In this stage it may be impossible to differentiate from a corneal scar due to other causes.

There was no pigment noted in the epithelium or in Bowman's membrane in any of the serial sections studied in our series that might correspond to a Fleischer's ring. As Terry and Chisholm[6] and Berliner[3] pointed out, Fleischer's ring, so impressive clinically by slitlamp microscopic examination, apparently has never been observed in any histologic preparation of keratoconus.

Stroma

The most striking feature in the stroma is an abnormal thinning and bulging forward of the central portion of the cornea (fig. 15). This thinning chiefly affects the stroma, which at the apex of the ectasia may be one third or less of its normal thickness. The lesions of Bowman's membrane as described previously, spread to the underlying stroma which shows areas of fibrillar degeneration and focal keratoblastic accumulation (figs. 4, 13, and 15). Eventually, the superficial stroma is destroyed and replaced by newly formed connective tissue in an irregular arrangement resulting in a wavy appearance (figs. 13 and 15). Apparently the pathologic process first is confined principally to the superficial stroma.

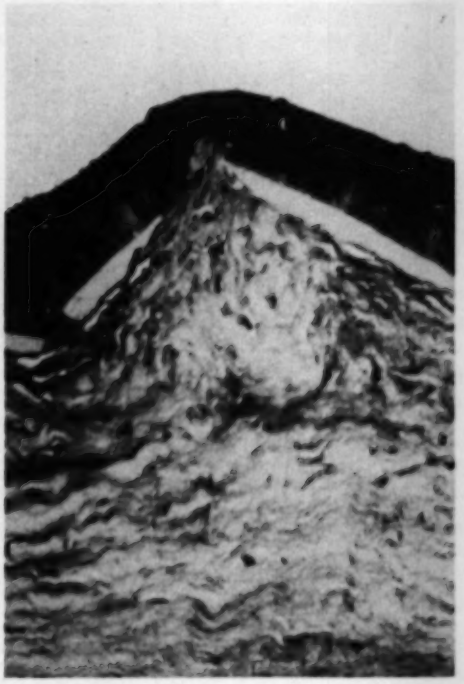

Fig. 13 (Chi, Katzin, and Teng). Two narrow gaps in Bowman's membrane filled by newly formed connective tissue. (×90, BP 98-1, toluidine blue stain.)

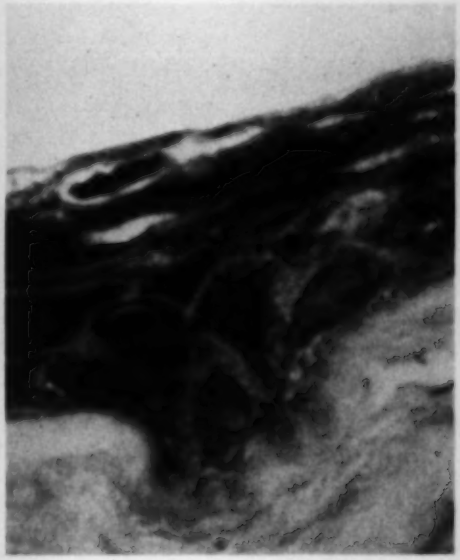

Fig. 14. (Chi, Katzin, and Teng). Gap in basement membrane and Bowman's membrane filled with epithelium. (×500, BP 100-7, periodic acid-Schiff stain.)

Histochemical tests with periodic acid-Schiff and the metachromatic stain of toluidine blue fail to demonstrate a difference in the concentration or distribution of the ground substance in any part of the corneal stroma in this stage. In advanced cases the stromal lesion extends more widely and deeply. In cases in which Descemet's membrane has been ruptured for some time, the edematous and degenerated stroma is re-

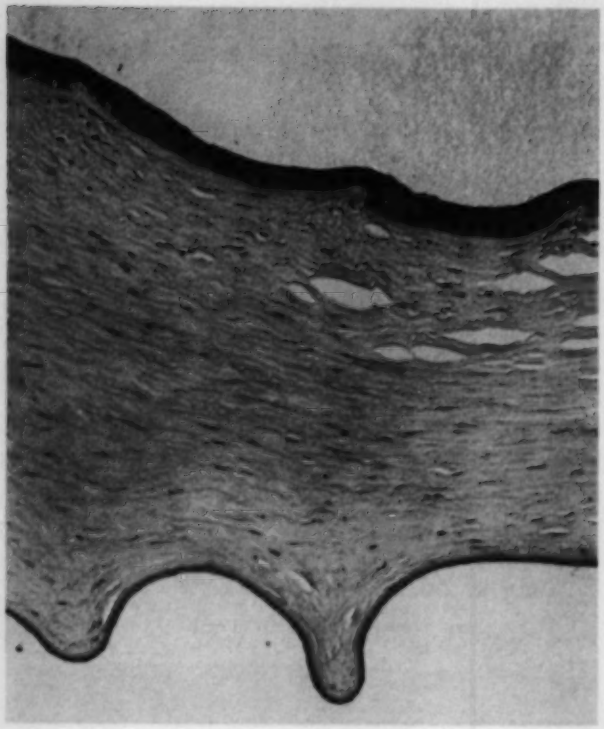

Fig. 15 (Chi, Katzin, and Teng). (1) Thinning of stroma at the apex of the cone. (2) Wavy appearance of Bowman's membrane with multiple narrow gaps filled with newly formed connective tissue. (3) Folds of Descemet's membrane and overlying stroma. (×100, BP 81-6, periodic acid-Schiff stain.)

paired by newly-formed scar and homogeneous masses are found, with loss of stromal cells (fig. 20). Deep stromal folds are always present if the sections are cut in a horizontal plane; that is, perpendicular to the deep striae which are seen as vertical corrugations in the slitlamp.

Descemet's membrane

Folds in Descemet's membrane associated with its overlying stroma are always present if the sections are cut in the right direction but no perceptible change in Descemet's membrane itself is observed in early stages (figs. 15, 16, 18, and 19). In our serial section study of the 65 cases there were eight (12.3 percent) with rupture of Descemet's membrane. The rupture involves the region of the greatest ectasia, near the center of the cone. Its extension varies. The edges of the rupture are curled anteriorly into the stroma (figs. 20 and 21). The defect in Descemet's membrane is first filled with endothelium, and later with a newly formed membrane which takes a paler stain with periodic acid-Schiff reagent (figs. 20 and 21).

Endothelium

The endothelium appears normal without appreciable change in early stages (figs. 16 and 17). But in the advanced stages the endothelium shows flattening and the nuclei are farther apart (figs. 18 and 19).

Comments

In view of the above description, the essential histopathologic findings in keratoconus in its early stages are chiefly confined to the anterior layers of the cornea. It involves the epithelium, the epithelial basement membrane, and Bowman's membrane,

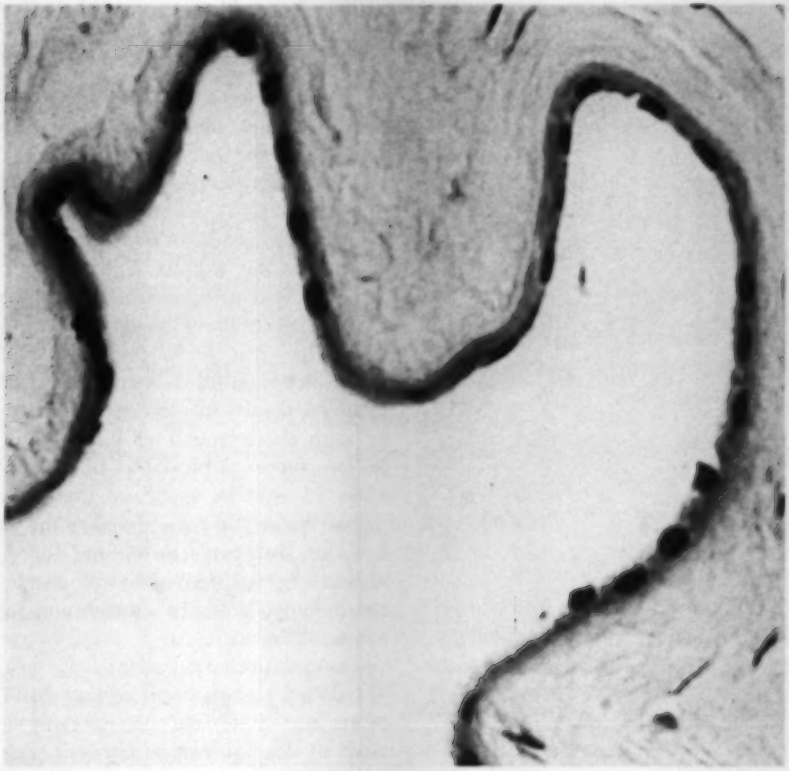

Fig. 16 (Chi, Katzin, and Teng). Folding of Descemet's membrane with nearly normal endothelium. (×210, BP 86-18, periodic acid-Schiff stain.)

as well as the anterior stroma. Changes in the epithelium seem to be secondary since they are found almost exclusively in the presence of changes in the basement membrane and Bowman's membrane, or changes in the endothelium. The degenerative changes in the basement membrane and Bowman's membrane are most likely the initial lesions, because they occur when the other layers of the cornea are normal. It is difficult or impossible to know whether the basement membrane or Bowman's membrane is first involved since their changes are always associated with each other.

Bowman's membrane is generally accepted to be a homogeneous, structureless membrane under ordinary light microscopic examination. Examination with the electron microscope, however, demonstrated that it consists of fine fibrils.[9] The first degenerative changes in Bowman's membrane appear to be swelling and fibrillation. It is quite possible that there is some physicochemical change in its constituents which give it the edematous and swollen appearance and make manifest its fibrillar structure which usually does not show under ordinary microscopy. This fibrillar degeneration extends to the underlying superficial stroma and can be demonstrated more clearly with the phase-contrast microscope. The overlying basement membrane first shows fragmentation, then complete destruction. It does not appear unreasonable to think, therefore, that the cornea becomes weakened by the degeneration of the membranes and superficial stroma,

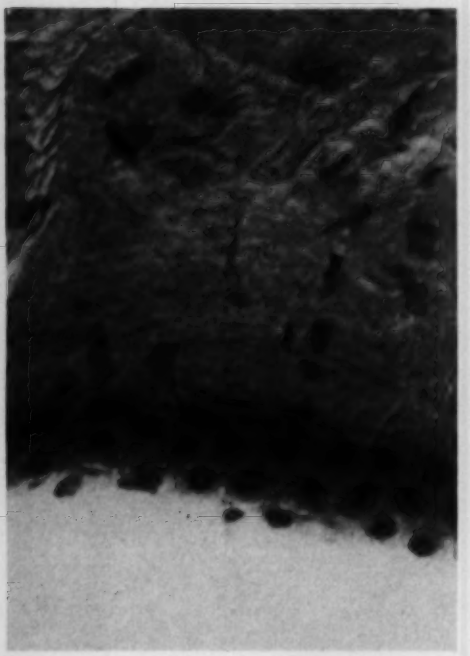

Fig. 17 (Chi, Katzin, and Teng). Endothelium seen in oblique section in keratoconus. No abnormality. (×140, BP 103-5, periodic acid-Schiff stain.)

and becomes unable to withstand the normal intraocular pressure. It cannot maintain its normal curvature and the result is conical cornea. At this stage, stretching of the cornea, ectasia, takes place without other obvious clinical signs.

It seems likely that the degeneration of the basement membrane and Bowman's membrane, as well as the anterior stroma, plays an important role in the development of keratoconus. As the degeneration develops, its manifestations progress from changes in the staining reactions to multiple areas of loss of Bowman's membrane and most of the superficial stroma. The gaps in the destroyed membrane are then filled with either newly formed connective tissue that contains focal concentration of keratoblasts or with downgrowth of epithelium. These lesions appear clinically as linear superficial scars. In addition, we believe that the stretching of the ectatic cornea widens the perforations of Bowman's membrane for the passage of nerves, and that this accounts for the holes in Bowman's membrane found in serial sections.

The questions may arise:

1. Would involvement of Bowman's membrane and superficial stroma alone be sufficient to develop conical cornea?

2. Is there any change in the stroma other than that in the anterior layers?

Regarding the first question, anatomically

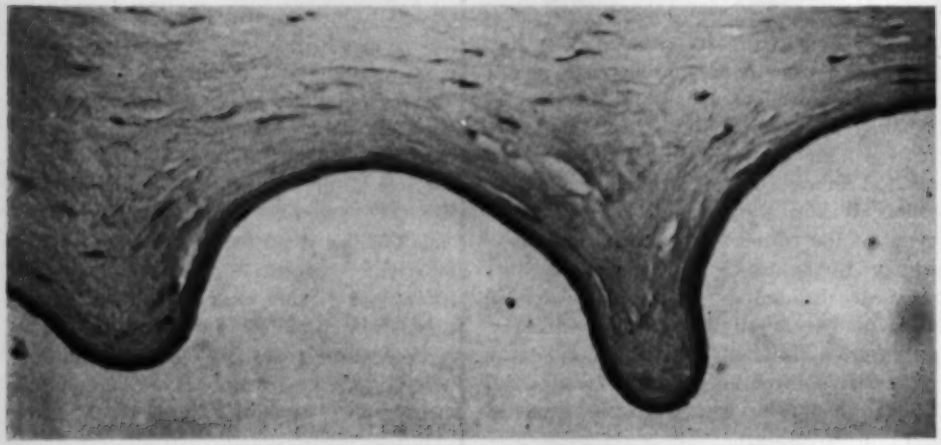

Fig. 18 (Chi, Katzin, and Teng). Folds in Descemet's membrane and deep stroma lined with flattened endothelium and widely spaced nuclei. (×112, BP 81-6, periodic acid-Schiff stain.)

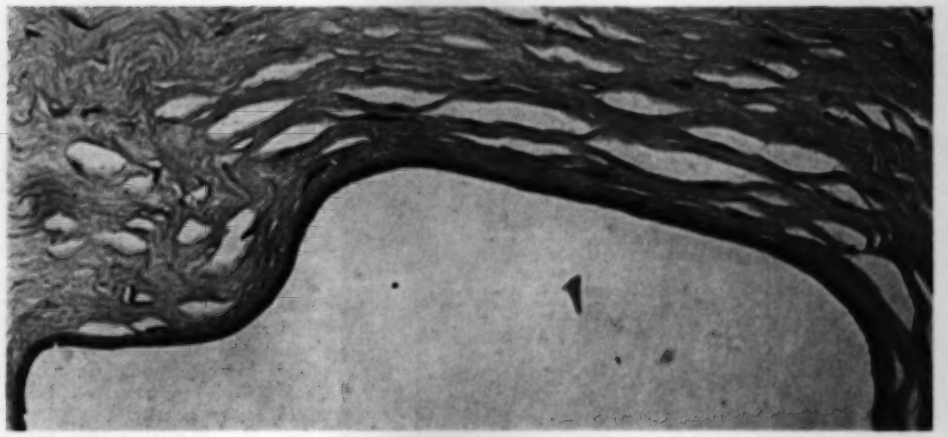

Fig. 19 (Chi, Katzin, and Teng). Folds of Descemet's membrane and deep stroma lined with flattened endothelial cells that are abnormally widely spaced. (×112, BP 86-6, periodic acid-Schiff stain.)

the anterior stromal layers are more compact and show more interlacing than the posterior layers.[10] In the opinion of Cogan,[11] the anterior compactness of the stroma serves to protect the curvature of the cornea. Clinically, in case reports of keratoconus posticus circumscriptus reported by Wise,[12] Leopold,[13] and Greene,[14] the cornea, at its thinnest portion, was reduced to one third or one fourth of its normal thickness and still withstood the normal intraocular pressure and maintained its normal anterior curvature. As Wise especially pointed out, the anterior cornea and its stroma were quite normal. Unfortunately, there was no histologic examination of these eyes.

One may ask, then, after a superficial keratectomy is performed, why does the cornea still retain its normal curvature? This is due to a replacement with fibrous tissue which reinforces the anterior cornea and gives it strength. Advantage was taken of this reaction on the part of the cornea in the treatment of keratoconus in the past. Cauterization and pressure bandages were used to promote fibrosis and restore the normal shape of the cornea.

Concerning the second question, relative to the posterior layer, reduction in the number and thinning of individual fibrils of the lamellae and changes in the ground substance and elastic fibers, all may be factors that contribute to the thinning and weakening of the stroma. The present methods of

Fig. 20 (Chi, Katzin, and Teng). (1) Rupture of Descemet's membrane showing: (A) Its edges are curled into the stroma. (B) The defect is filled by regenerated endothelium and newly-formed membrane. (2) Hyaline degeneration of the anterior two thirds of the stroma. (3) Irregularity of the thickness of the epithelium. (×100, BP 99-2, periodic acid-Schiff stain.)

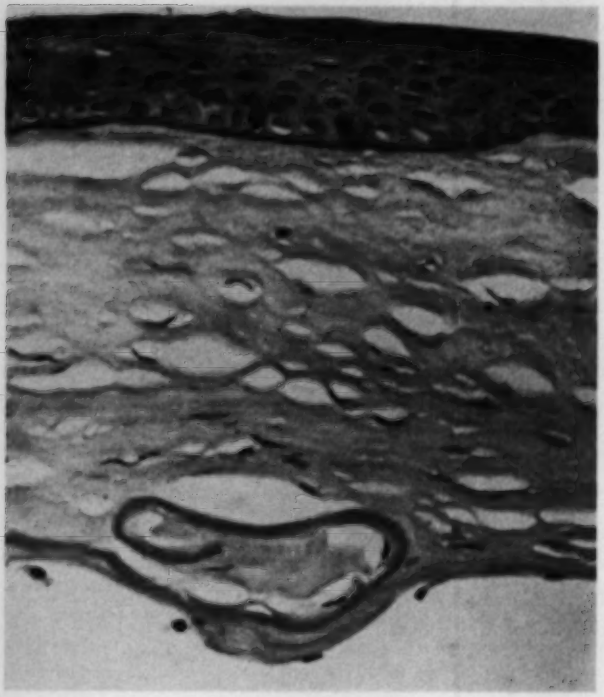

Fig. 21 (Chi, Katzin, and Teng). Rupture in Descemet's membrane showing the edge curled anteriorly into the stroma. Poorly developed newly formed Descemet's membrane is present in the gap. (×120, BP 99-7, periodic acid-Schiff stain.)

study do not show perceptible change in the rest of the stroma in the early stages. It is impossible to count the number of the fibrils of the lamellae and measure their thickness.

Detailed comparative studies of the character of the individual corneal fibrils in normal and keratoconus eyes with electron microscope technique are contemplated. The ground substance of the corneal stroma may be demonstrated by periodic acid-Schiff stain and metachromatic stain with toluidine blue. There was neither increased nor decreased metachromatic staining, nor any change in intensity of stain in periodic acid-Schiff.

So far our admittedly imperfect technique of studying the histochemistry of the stroma shows that there is no change in the ground substance of the rest of the stroma in the early stages of the disease. Verhoeff's elastic fiber method failed to demonstrate the elastic fibers in the cornea, although it did demonstrate the elastic fibers of the sclera very satisfactorily. The elastic fibers of the cornea are very difficult to study. It has not been possible to draw definite conclusions regarding any change in the elastic fiber content of the cornea.

Folds in the deep stroma and in Descemet's membrane are a common finding in our series. Clinically these are seen as the stromal striae in the deep layers of the cornea. We believe that the deep folds and buckles of Descemet's membrane are emphasized by release of tension after the removal of the disc.

Von der Heydt[15] had observed clinically a very decided change in size and shape of these striae at various times. Schmidt[16] stated that the striae in keratoconus may disappear transiently with a temporary increase in the intraocular pressure, through compression of the globe, abdominal pressure, and so on. It was his opinion that the striae in keratoconus were caused by the contraction force of

Descemet's membrane.

In order to exclude the possibility of artefact produced through histologic preparation, we studied a number of discs with other pathologic processes, such as Fuchs' dystrophy and corneal scars due to other causes. They did not show folds in the stroma and Descemet's membrane.

Eight out of 65 cases (12.3 percent) showed rupture of Descemet's membrane. According to a study by Terry and Chisholm[8] it appeared that strips of rabbit cornea could be stretched some 10 percent before they ruptured. Therefore, any overstretching may sufficiently disrupt the continuity of the membranes of the cornea in keratoconus. Hervouët found one out of nine cases with rupture of Descemet's membrane in histologic examination. Clinically Rohrschneider and Stohr[17] reported that it occurred in about 11 percent of all cases of keratoconus. One would expect higher percentages in specimens that come to pathologic examination, because this would not include the earliest cases.

Since the endothelium is intimately associated with Descemet's membrane, its cells are lengthened and flattened, and the nucleus is reduced to a thin dark particle.

Summary

A type of degeneration is found in the basement membrane, Bowman's membrane, and the anterior stroma of the cornea in keratoconus. The mechanism of development of keratoconus, in our opinion, is due to pathologic processes with subsequent weakness of the cornea which becomes unable to withstand the normal intraocular pressure, resulting in bulging of the cornea. The ectatic cornea is stretched and appears thinner than normal. The cause of the degeneration remains unknown. It begins in Bowman's membrane or the basement membrane of the epithelium and spreads to the adjacent superficial stroma. The nature of the degeneration is not entirely clear but fibrillation and the change in staining properties of Bowman's membrane itself are shown by an alteration in their reaction with periodic acid-Schiff and toluidine blue stain. As the pathologic processes continue, the basement membrane and Bowman's membrane, as well as the anterior stroma, show actual destruction in places and new-formed connective tissue fills these gaps. In more advanced cases Descemet's membrane eventually ruptures and the whole thickness of the cornea is involved.

Conclusions

The essential pathologic process of keratoconus is a degenerative lesion chiefly involving the anterior layers of the cornea. The striking common characteristic histologic findings are:

1. Fragmentation of the basement membrane of the corneal epithelium, fibrillation of Bowman's membrane, and the anterior stroma in the early stages.
2. Marked thinning and bulging of the central cornea.
3. Wavy appearance of Bowman's membrane with multiple narrow gaps filled either by newly formed connective tissue that contains focal accumulations of keratoblasts or by epithelium.
4. Folds and buckles in Descemet's membrane and its overlying deep stroma.
5. Rupture of Descemet's membrane in 12.3 percent of our series.

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THE TECHNIQUE OF LAMELLAR KERATECTOMY

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There are three surgical procedures to improve vision due to scarring of the central portion of the cornea: penetrating keratoplasty, lamellar keratoplasty, and lamellar keratectomy, or a combination of these methods. It has been known for several decades that penetrating keratoplasty in a densely opaque cornea is followed by opacification of the transplant. The same poor prognosis is true when it is done in cases of various pathologic conditions of the cornea, such as opacities following blast injury or severe chemical burns. Furthermore, this operation may end, in a not negligible percentage, with poorer than the preoperative vision. These considerations broaden the indications of lamellar keratoplasty and keratectomy. There are, as yet, no sharp dividing lines between the indications for these three methods and it is not the purpose of this paper to discuss this problem.

A basic prerequisite of a successful lamellar keratoplasty is the transparency of the deepest layers of the stroma. The same holds for lamellar keratectomy. To get some visual benefit the deepest corneal layers must be at least moderately transparent. Though lamellar keratectomy is the safest of the three operations, it will yield the least spectacular visual improvement. Whether it is an operation per se or the first step to lamellar keratoplasty, it has to fulfill the following conditions in order to get the best possible results:

a. As much cornea should be removed as is feasible. Not more than one or two tenths of a mm. should be left on Descemet's membrane. It is risky to try to cut that deep with the trephine.

b. The removal should be carried out in one and the same cleavage plane in order to cause the least trauma to the stromal fibers. The more damage to the fibers the more scar tissue will be produced and new vessels will enter the cornea. It is questionable that a sharp instrument can be advanced in the same cleavage plane.

c. The prepupillary area should be

handled with the greatest care. In case the dissection of the lamella starts from the center, this area suffers the most trauma.

Technique

A technique fulfilling these conditions has been used by me for several years. Lamellar keratectomy belongs in the realm of microsurgery. Therefore good illumination and magnification are essential. Preoperatively, antibiotics are given locally and, if there is no contraindication to mydriatics, the pupil is dilated. The dark background facilitates work on the not fully opaque cornea. Drop and retrobulbar anesthesia is used.

The eyeball is fixed at two points of the limbus. A broad keratome is introduced tangentially at the limbus, or even slightly in the sclera, corresponding to the clearest portion of the peripheral cornea. The tip of the keratome is advanced about three mm. to the deepest layer of the cornea, and held in this position. Now, about one mm. back of the tip, a Tooke knife is used to cut down vertically to the keratome (fig. 1) the wound being made approximately parallel to the limbus.

The keratome is removed and a narrow, curved corneal splitter (fig. 2) is used to start the actual lamellar splitting by pressing it against the peripheral lip of the wound made with the Tooke knife. The tip of the splitter is advanced into the base, that is in the deepest portion of the central lip of the wound. It is moved forward and sideways until a large enough area of the cornea is undermined to give room to a small Gill knife (fig. 2). The whole cornea is undermined in the same cleavage plane except for the one- to two-mm. peripheral portion. This maneuver is a splitting not a cutting of the cornea, which a sharp instrument unavoidably would do. To facilitate the advance of the Gill knife, an incision is made in the undermined cornea. In this way the undermined portion can be lifted.

After the undermining is completed, the

Fig. 1 (de Roetth). Broad, reflex-free keratome in place. The tip cannot be seen. Lower edge of keratome is covered by the Tooke knife. This cuts deep, to the tip of the keratome.

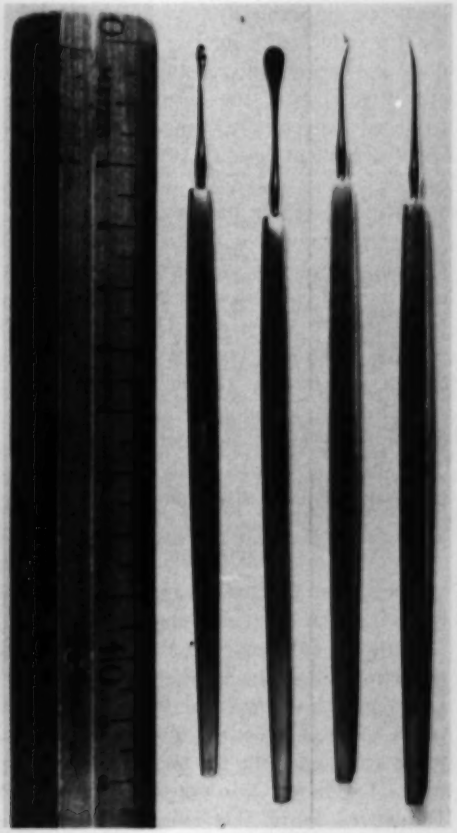

Fig. 2 (de Roetth). Microsplitter and small Gill knife. The curvature of their blades approximates that of the cornea.

cornea is severed with corneal scissors (Castroviejo's or Katzin's) one mm. from the limbus. In lamellar keratoplasty, the same technique can be used by changing the place of entrance of the broad keratome according to the size of transplant to be used.

Difficulty may arise when the entire cornea is densely scarred, in which case the tip of the keratome cannot be seen. However, in most cases of corneal scarring the periphery is somewhat translucent, or there is a small portion of the periphery not entirely opaque. The keratome can be applied in this segment. It also may happen that, while attempting to advance the tip of the keratome into the deepest corneal layers, the tip enters the anterior chamber. This does not necessitate postponement of the operation because the keratome, being introduced tangentially, can be pulled back slightly without loss of aqueous. It again can be advanced a little more tangentially and stopped before perforation occurs.

The postoperative treatment consists in applying antibiotic ointment twice a day until corneal epithelization is complete. This takes four to seven days. Irradiation with X rays or radium is necessary only if the cornea was heavily vascularized. After the epithelization is complete, there usually is no or only slight pericorneal congestion. This can be treated with locally applied cortisone but only after complete epithelization.

Discussion

There is no uniformity among the authors in their techniques of doing lamellar keratectomy or in removing the opaque discs preliminary to lamellar keratoplasty. Paufique[1] uses his angulated knife to get the plane of cleavage after outlining the area to be removed with the trephine, and continues the dissection of the cornea with the Desmarres knife. The same technique is followed by Paton[2] and by A. O. Leigh.[3]

Castroviejo[4] is using his special trephine with micrometer regulation to outline the area to be excised. The dissection of the leukoma is started with a special knife made with razor blade material and he also recommends a cataract or Lundsgaard or Desmarres knife for this purpose. The excision is continued with blunt scissors in the same plane of cleavage. The reason for using blunt scissors is to minimize the tendency to perforate the cornea. "This often occurs when only a knife is used, particularly in dissecting deep opacities in the vicinity of Descemet's membrane."

Rycroft and Romanes,[5] when doing lamellar grafts, remove the corneal disc with the help of a trephine and a miniature Desmarres blade. "The disc should be separated by smooth strokes rather than by small irregular cuts. . . . The disc should be removed in one piece of even thickness, but when the scar is soft and pliable, this is not always possible."

Perritt[6] starts the removal of the corneal lamella with a cruciate incision which is Wiener's original method. The actual removal is started from the apex of the quadrant, that is in the center of the cornea, with a safety razor blade, carefully staying in the same plane of cleavage.

Shafer[7] in experimental surgery on rabbits removed the lamella with a Bard-Parker knife in the same cleavage plane. Only Stansbury[8] applies a dull instrument, an iris repositor, after outlining the area with a deep cut into the stroma. In this way he stays in the same cleavage plane.

Summary

The indications of lamellar keratectomy and lamellar keratoplasty seem to expand at the expense of penetrating keratoplasty. To get the best visual results it is imperative that the removal of the corneal lamella be performed in the deepest corneal layers and in the same cleavage plane. A safe operative technique is described.

Old National Bank Building (1).

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READING AIDS FOR THE PARTIALLY BLIND*

New methods of rating and prescribing optical aids

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Introduction

A majority of the optical aids for reading are based on the simple principle that the retinal image increases in size as the object is brought closer to the eye. The function of such a device is to permit the object to be viewed from a short distance without making excessive demands upon accommodation. In its simplest form this type of magnifier is a convex lens which may be worn as a spectacle, held in the hand, or supported in a mount which rests on the reading page.

It is important to prescribe the minimal magnification required by the particular patient because of the limitation of field, decreased working distance, distortion, and other aberrations which accompany high magnification. It is not always recognized that the minimal dioptric power of the magnifier which is needed to give useful reading vision depends not only upon the patient's visual acuity but also upon the closest distance for which he can accommodate *without* the magnifier.

The specified magnifying powers of most of the commercially available reading aids are computed from the simple formula, $M = D/4$, in which D is the dioptric power. This conventional formula makes the arbitrary assumption that with the unaided eye all patients can sustain just enough accommodation to hold the reading matter at a distance of 25 cm. It assumes also that, when the magnifier is used, the reading material is located in the principal focal plane of its lens, so that no accommodation is required of the emmetropic eye. Since neither of these assumptions applies in a majority of cases, the conventional ratings of magnification are of limited use in the selection of a reading aid for a given patient.

Consider for example three patients each of whom can just read, at 40 cm., print which is four times the size of newsprint.

The first patient is an emmetrope who can sustain not more than three diopters of accommodation. With a +7.0D. reading glass, either worn as a spectacle or held close to the eye, he will be able to read newsprint which is held at about 10 cm. from the lens. When the object is at this distance the light rays leave the 7.0-diop-

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ter lens with a divergence of three diopters.

The second patient has a myopia of two diopters and can also sustain three diopters of accommodation. Without a correction for his myopia he needs only a five-diopter magnifying lens to read newsprint. As before the print must be 10 cm. from the lens.

The third patient is a child with one diopter of hyperopia who can sustain 11 diopters of accommodation. He must also hold newsprint at a distance of 10 cm. from the eye, but he needs no optical aid to read at this distance because of his high power of accommodation.

The purpose of the present study is to outline procedures for selecting, with the minimal amount of trial and error, the optical aid best suited for each individual, as determined by the dioptric power of the device, by the patient's visual acuity, and by his near-point of accommodation. The detailed application of these procedures to the use of the many types of optical reading aid, and the evaluation of the results obtained will be reported in subsequent papers.

The procedures may be divided into three steps:

Step 1 is the development of formulas which specify, for the various types of optical aid, the size of the retinal image relative to its size when viewed without the magnifier from the normal reading distance of 40 cm. These formulas take account of the fact that the magnification depends not only on the dioptric power of the magnifier itself, but also on the diopters contributed by the observing eye when using the magnifier.

Step 2 involves a determination of the closest distance, expressed in diopters, at which the patient can sustain sufficient accommodation for reading.

Step 3 consists of a determination of the smallest size of print which the patient can read at 40 cm. This measurement determines the factor by which the retinal image must be increased to permit the reading of ordinary newsprint.

1. Formulas for computing magnification

Our method of rating the magnifying powers of optical aids for near vision differs in two respects from the conventional ratings based on the formula $M = D/4$. First, the conventional formula measures the size of the retinal image relative to its size when the same object is viewed from a distance of 25 cm. Our method specifies the magnification relative to a distance of 40 cm., in order to correspond to the distance at which we determine the acuity. The symbol M_{40} will hereafter be used to designate this particular measure of relative magnification. Secondly, the conventional formula applies only to the special case in which the object viewed is located at the anterior principal focus of the lens. Our formulas take account of the fact that, unless the object distance is preset at the principal focal distance of the magnifier, many patients can obtain a further increase in the size of the retinal image by bringing the print closer to the magnifying lens.

The derivations of the formulas are given in Appendix 1. Their meaning and use can be illustrated by calculation of the magnifying powers of several representative forms of optical aid. For simplicity in this discussion we shall assume that the magnifier is a simple thin convex lens.

A. Magnifiers in which the distance from object to lens can be varied

This group includes the following types: (1) A convex lens worn as a spectacle; (2) a hand-held reading glass; (3) a focusable device, consisting of a convex lens, a support which holds it at a preselected distance from the reading page, and a means of adjusting this distance.

Let us assume for purposes of illustration that an emmetropic patient is given a +20D. lens in the form of a spectacle. If he holds the print at a distance of five cm. from the lens, the emerging light will have zero ver-

gence. With relaxed accommodation the patient therefore obtains a clearly focused retinal image. The size of the retinal image is eight times that formed when the print is viewed from 40 cm. without the lens. The magnification, M_{40}, is therefore $\frac{20}{2.5}$ or 8.

If, however, this patient can sustain three diopters of accommodation, he can obtain a greater magnification by bringing the print to a distance of 1/23 meter (43.5 mm.) from the lens. The divergence of light incident on the lens is now 23 diopters, the divergence of light leaving the lens is 23 − 20 or three diopters. The magnification in this case is $\frac{20 + 3}{2.5}$ or 9.2.

The general formula may be written

$$M_{40} = \frac{D + A}{2.5} \qquad (1)$$

in which D equals the dioptric power of the magnifier and A equals the diopters contributed by the observer's own eye when using the magnifier. For the emmetrope (or the corrected ametrope) A equals the accommodation exerted by the observer when using the magnifier.

Formula (1) assumes that the observer's eye is close to the lens. If the lens-to-eye distance is appreciable the magnification is given by the formula

$$M_{40} = \frac{D + A - hAD}{2.5} \qquad (2)$$

in which h is the lens-to-eye distance expressed in meters. Suppose for example that a 20 diopter reading glass is held 166 mm. from the eye and 1/26 meter (3.84 mm.) from the print. A virtual image is formed by the lens, 167 + 166 mm. or one-third meter from the eye. The emmetrope must therefore exert three diopters of accommodation in order to obtain a clearly focused retinal image. From formula (2) the magnification of the retinal image is

$$\frac{20 + 3 - .166\,(60)}{2.5} \text{ or } 5.2$$

When the object is located at 1/23 meter (43.5 mm.) from the lens and the eye is close to the lens the same three diopters of accommodation are required but, since h in this case is zero, the magnification is

$$\frac{20 + 3}{2.5} \text{ or } 9.2$$

These examples demonstrate the two conditions that must be fulfilled if the observer is to obtain the greatest possible magnification from a focusable optical aid:

The first requirement is that the eye be close to the lens.

The second requirement is that the reading matter be as close to the lens as the observer's power of accommodation will permit.

There is, however, one special situation in which the magnification is independent of h, the lens-to-eye distance. When the object is located in the anterior principal focal plane of the lens, the rays leaving the lens have zero vergence, the virtual image of the object is at infinity, and no accommodation is required of the emmetropic eye (A = 0). In this case the magnification is not decreased as the eye is withdrawn from the lens. Formula (2) shows that in this case M is independent of h, since, when A is zero, the term A − hAD in the numerator is zero for all values of h.

There is, however, a *spurious* impression of increased magnification as the eye is moved away from the lens. This is because the retinal image of the lens aperture decreases in size with increase in its distance from the eye. When compared with the apparent size of this aperture, the image of the print seems to become larger as the eye is withdrawn. The number of letters which can be seen at one time through the lens aperture therefore decreases with in-

crease in the distance of the eye from the lens. The aberrations also increase because light from the more peripheral portions of the lens enters the pupil. Consequently even when the object is in the principal plane of the lens, and the size of the retinal image is therefore independent of the eye-to-lens distance, the eye should nevertheless be close to the lens in order to reduce the aberrations and to obtain the widest possible field of view.

B. Magnifiers in which the distance from object to lens is fixed

In some of the magnifiers which rest on the reading page the distance of the lens from the base of the support is not adjustable. This predetermined distance is in certain devices nearly equal to the principal focal length of the lens. In others it is much less than the focal length.

If, for example, the print is located at a fixed distance of 38.4 mm. from a 20-diopter lens, the light emerges with a divergence of six diopters and the virtual image lies 167 mm. behind the lens. The observer who can accommodate for six diopters of divergence can place his eye close to the lens. If he can accommodate for a divergence of only three diopters, he must place his eye 333 mm. from the virtual image, 333 − 167 or 166 mm. from the lens.

Formula (2) shows that in the first case, in which h equals zero and A equals six diopters, M_{40} equals $\frac{20+6}{2.5}$ or 10.4. In the second case M_{40} equals

$$\frac{20+3-0.166\ (60)}{2.5} \text{ or } 5.2$$

This example shows that, as was the case with focusable magnifiers, the observer obtains the greatest benefit from a given device when he brings his eye as close to its lens as his power of accommodation will permit. In nonfocusable magnifiers in which the fixed object-to-lens distance is less than the principal focal length of the lens, the minimal eye-to-lens distance is determined by the power of accommodation which can be sustained. The quantities h and A in formula (2) are in such cases not independent. A more convenient expression for computing the magnification when u, the object-to-lens distance, is fixed and h depends upon A is

$$M_{40} = \frac{1}{1-Du}\ \frac{A}{2.5} \qquad (3)$$

In the example previously mentioned the factor

$$\frac{1}{1-Du} = \frac{1}{1-20\ (.0384)} = 4.3$$

so that for this magnifier

$$M_{40} = \frac{4.3A}{2.5}$$

For the observer who can overcome six diopters of divergence M_{40} equals 10.4. For the observer who can overcome only three diopters of divergence, M_{40} equals 5.2. Formula (3) gives therefore the same values for the magnification as were obtained previously by applying formula 2.*

C. Projection magnifiers

These devices differ from those previously discussed in that the observer views, not a virtual image, but a real enlarged image which is formed on a screen. Let us assume that the projected image is five times the size of the actual reading matter. If the observer views the screen from a distance of 40 cm., for example, there is a fivefold increase in the retinal image as compared to its size when the print is viewed directly from the same distance of 40 cm. If however the observer views the projected image from a distance of 20 cm. there is a 10-fold

* It should be noted that in the special case in which u equals 1/D, that is, when the object is in the principal focal plane of the lens, 1-Du and A are both equal to zero and formula (3) leads to an indeterminate value for M. To compute the magnification in this special case formula (2) must be used, and since A equals zero this formula reduces to $M_\infty = D/2.5$.

increase in the retinal image. As in the previous cases, therefore, the size of the retinal image which a given patient can obtain depends not only on the magnification of the device itself but also on A, the dioptric equivalent of his near point. A general formula applicable to this type of device is

$$M_{40} = M_p \frac{A}{2.50} \qquad (4)$$

in which M_p is the ratio of the size of the projected real image to the size of the object.

D. Application of the formulas for magnification

The applications of the different formulas can be illustrated by showing how they are used to compute the magnifications of representative types of reading aid. Those we have chosen for purposes of illustration are selected from the optical reading aids which can be purchased from the American Foundation for the Blind, Department of Special Services. Data on these and other magnifiers are tabulated in Appendix 2.

1. *Adisco illuminated magnifier.* This device utilizes a doublet lens whose equivalent power is about 21 diopters. The lens is held in a fixed mount at a distance from the reading page only slightly less than the equivalent focal length of the lens system. The rays of light therefore emerge with such slight divergence that A can be taken as equal to zero. By formula (1),

$$M_{40} = \frac{21}{2.5} \quad \text{or} \quad 8.4$$

In using devices of this type the patient should wear whatever spectacles he may require for clear vision at distance. The presbyope, for example, must be cautioned to look through the distance portion of his bifocal spectacles and may find that the magnifier is easier to use in conjunction with single vision lenses correcting his refractive error for distance.

2. *Plasta cataract reader.* In this device a lens of about 17 diopters is supported in a mount which locates the reading page at a fixed distance of about 40 mm. from the lens. The rays of light therefore leave the lens with a divergence of 25 − 17 or eight diopters, and form a virtual image at a distance of 12.5 cm. behind the lens.

An emmetrope who can sustain eight diopters of accommodation can hold his eye close to the magnifier. The emmetropic presbyope who can sustain only one diopter of accommodation must place his eye one meter from the virtual image, that is, 87.5 cm. from the lens. If, however, he wears a three-diopter reading spectacle he can overcome three diopters of divergence and can locate his eye at 33 − 12.5, or 20.5 cm., from the magnifier lens. The magnification obtained in each case can be computed from formula (2), or more easily from formula (3). According to the latter formula, for this magnifier

$$M_{40} = 3.1 \frac{A}{2.5}$$

Since A is eight diopters in the first example, one diopter in the second, and three diopters in the third, the magnifications are respectively 9.9, 1.24, and 3.72. For the patient who requires only a threefold or fourfold increase in size of the retinal image this reading aid has the advantage that the eyes may be at a comfortable viewing distance from the magnifier lens. It is important to remember, however, that the virtual image is only 12.5 cm. behind the lens and the presbyope must therefore wear a suitable reading spectacle.

3. *Tripod magnifier.* This device has a 28-diopter doublet lens supported in a threaded mount which permits variation of its distance from the reading page. When focused for the shortest object-to-lens distance, the light emerges with a divergence of about eight diopters. When the reading material is at the greatest object-to-lens distance, the light leaves the lens with a convergence of

about two diopters. Patients with uncorrected spherical errors ranging from two diopters of hyperopia to eight diopters of myopia can therefore adjust this magnifier so as to obtain a clearly focused retinal image.

The emmetrope who can sustain as much as eight diopters of accommodation can use any object-to-lens distance from that giving zero vergence to that giving eight diopters of divergence. The presbyope can focus it for use with either his correction for distance or his reading spectacle.

To obtain the greatest possible magnification the patient must place his eye close to the lens and must use the shortest object-to-lens distance that his accommodation will permit. When h is zero formula (2) applies and shows that, for this magnifier, M_{40} varies from 11.2 for $A = 0$ to a maximum of 14.4 when $A = 8$ diopters.

Of the several types of reading aid the focusable magnifiers are the most versatile. When the location of the virtual image can be varied over a wide range, spectacle correction of any spherical error of refraction is unnecessary. This range of adjustment, moreover, permits full use of the observer's power of accommodation in obtaining the necessary increase in size of the retinal image.

2. Measurement of near-acuity as a basis for selection of reading aid

In many cases the size of print which the patient can read at a given near distance can be predicted with fair accuracy from conventional measurements of acuity made with isolated letters at a distance of 20 feet. In some situations, however, there are significant differences in the acuities under these two conditions.

Patients with central lens opacities, for example, may have lower acuity for near than for distant objects, those with peripheral lens opacities a higher acuity, because of a decrease in diameter of the pupil in near vision. In such cases a measurement of near-acuity gives more valid data for the selection of an optical aid for reading.

A determination of near-acuity, if based on ability to read textual material, is preferable to measurements with isolated letters because it reproduces more closely the actual situation in which the optical aid will be used. Of two patients with the same acuity for isolated letters, the rapid reader, who does not need to see clearly each letter or even each word, may prefer a lower magnification because of the associated wider field. The slow reader may require a higher magnification and, just because he reads slowly, may not object to a restricted field of vision. Measurements based on the smallest size of type which the patient finds adequate for reading at a fixed distance take account of such individual differences in ease of reading.

As a basis for the selection of a reading aid we therefore use continuous sentences in a graded series of sizes viewed from a fixed distance of 40 cm. The most useful specification of their sizes is one based on the metric notation. This gives the distance in meters at which the height of the lower case letters subtends a visual angle of five minutes.

As compared with the Jaeger designations, a specification of print sizes in M units has the advantage that the size of the print is directly proportional to its M value. For example, 5M print is five times as large as 1M print. The numerical designations of the Jaeger types have no such quantitative relationship to the sizes of the print.

Conversion of acuity measured in M units to the Snellen notation and vice versa is simple, since the M value specifies the distance in meters at which the letters subtend a visual angle of five minutes. If for example 2M is the smallest print which the patient can read at 40 cm., then his near-acuity in the Snellen notation is $\frac{40}{200}$. The

equivalent distance acuity is $\frac{20}{100}$. Conversely if the distance acuity is, for example, 10/250, the equivalent near acuity is $\frac{40}{1,000}$, which corresponds to ability to read 10M print at 40 cm. It should be emphasized however that because of the factors previously mentioned the actual acuities may not be the same when measured at 20 feet with isolated capital letters and at 40 cm. with textual material.

A further advantage of the metric notation is that ordinary newsprint is of approximately the same size as standard 1M print. The patient who can read print of this size at the usual reading distance of 40 cm. will therefore ordinarily not require any special optical aid other than that which would normally be prescribed for reading at this distance. If on the other hand 4M print is the smallest that can be read at 40 cm. it is obvious that to read newsprint the patient requires about a fourfold increase in the size of the retinal image. The measurement in M units of the smallest print which can be read at 40 cm. gives therefore a direct measure of the required magnification of the retinal image. Our complete series of test cards comprises textual matter of the following sizes in M units: 1.0, 1.25, 1.50, 2, 3, 4, 5, 6, 7, 8, 9, and 10M.

The test of near-acuity provides one of the two essential items of information needed to select the proper optical aid for reading and gives a direct measure of M_{40}, the magnification of the retinal image required to permit the reading of 1M print. The second important item of information concerns the accommodative power.

3. Measurement of the near-point of accommodation

To select the minimal dioptric power of the optical aid which may be expected to provide useful reading vision, it is necessary to know the shortest distance for which the patient can sustain accommodation. The reciprocal of this distance, expressed in meters, gives the numerical value of A to be substituted in the formula relating magnification to the dioptric power of the optical aid. It is not easy to obtain precise and reliable measures of the near-points of accommodation of patients having varying degrees of subnormal acuity. The size of the test target used for each patient must be commensurate with his visual acuity. The conventional method of measuring the near-point may give misleading information because of the increase in size of the retinal image as the test target is brought closer to the eye. Because of increased visibility associated with a larger retinal image, the decrease in visibility associated with an out-of-focus image may not be noticed by the patient with a very low acuity.

We have devised a simple instrument (fig. 1) which eliminates this difficulty and provides fairly reproducible measures of the near-point of accommodation even when the acuity is markedly reduced. Based on the principle of the Badal optometer,[2] it provides for any desired change in the apparent or optical distance of the test object without associated change in the size of the retinal image.

Our instrument uses a 16-diopter lens, L in Figure 1, located at a fixed distance from the observing eye. This distance is so chosen that the posterior principal focal plane of the optometer lens coincides approximately with the anterior focal plane of the eye. When the test target, shown at T in Figure 1, is at the zero point of the optometer scale, 62.5 mm. from the lens, the rays of light reach the eye with zero vergence. Each decrease of four mm. in distance of the target produces an increase of divergence of one diopter at the anterior focal plane of the eye. As the target is brought nearer, therefore, there is a gradual increase in the diopters of accommodation required, but no increase in the size of the retinal image. Since the spectacle plane is within a few

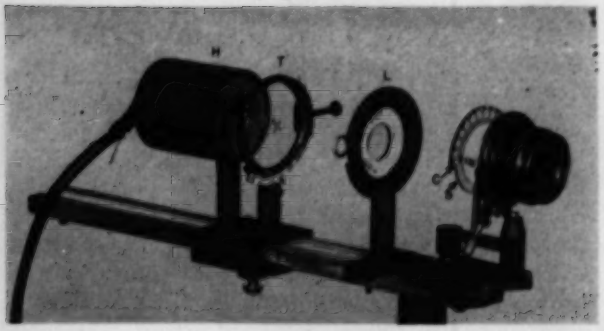

Fig. 1 (Sloan and Habel). Optometer for measuring the near-point of accommodation.

millimeters of the anterior focal plane, diopters of accommodation as measured by the optometer are equal to the diopters of spectacle power needed to overcome the same divergence.

Spheres and cylinders may be inserted before the observing eye, at S and C, to correct the patient's error of refraction. When the ammetropia is corrected the near-point reading in diopters gives a direct measure of the total range of accommodation. The accommodation that can be sustained is, according to accepted practice, one half to two thirds of the total range. For the emmetrope or the corrected ametrope, this is the value substituted for A in the magnification formulas.*

The test target T in Figure 1 is located in front of a diffusing glass illuminated by means of a six-volt lamp, enclosed in a housing, H. Further investigation is needed to select the best form of target for measuring the near-points of patients with varying degrees of vision. At the present time we are using two different types of target. The first is the simple crossline pattern shown in the photograph. When viewed in the instrument the two arms of the cross subtend visual angles of six and seven degrees. For patients whose acuity is 20/100 or better a target is used in which the component lines have a width of about five minutes. If the patient's acuity is less than 20/100 a target composed of heavier lines, subtending a visual angle of about 20 minutes, is used.

The second group of targets consists of Landolt rings of 12 different sizes. The acuities required to locate the gaps in the rings range from 0.025 (16M at 40 cm., 40/1,600, 5/200) for the largest to 0.5 (0.8M at 40 cm., 40/80, 20/40) for the smallest. The near-point is measured with the smallest target which the patient can see when it is clearly focused. For all levels of acuity therefore it is possible to measure the dioptric distance at which insufficient power of accommodation results in a significant impairment of acuity.

It should be recognized, however, that the measured near-points of patients with markedly subnormal vision may not necessarily represent accurately their true physiologic powers of accommodation. The patient with very low acuity may not perceive the first stages of blurring of the retinal image and his true power of accommodation may therefore be overestimated. It is possible on the other hand that, when acuity is markedly reduced, the normal stimulus to accommodation may be weakened and the true power of

* Reference to the derivation of the formulas given in Appendix 1 will show that when A, the dioptric power contributed by the observing eye, is measured in the anterior focal plane and when the magnifying lens D is actually located in this plane then the magnification of the retinal image is proportional to $D + A$. When the lens is at a distance h (in meters) from the anterior focal plane the magnification is proportional to $D + A - hAD$. This will be recognized as the formula for the equivalent dioptric power of two lenses, D and A, separated by an interval h.

accommodation therefore underestimated. The measures of accommodation provided by the optometer are nevertheless of value in prescribing an optical aid for reading. For this particular purpose it is necessary to know only the *apparent* range of accommodation as determined by the dioptric distance at which there is a noticeable impairment of acuity because the accommodative response is insufficient.

Summary

A procedure is outlined for selecting, with a minimal amount of trial and error, the weakest magnifier which will give useful reading vision to the patient with subnormal acuity. It is shown that selection of the proper reading aid depends not only upon the visual acuity but also upon the near-point of accommodation.

The testing procedure involves (a) measurement of the smallest size of print which the patient can read at a standard distance of 40 cm., and (b) measurement in diopters of the closest distance for which the patient can accommodate. Formulas are given for rating the magnifications of various forms of optical reading aids which take account of the fact that the increase in size of the retinal image varies not only with the dioptric power of the magnifier itself but also with the diopters of accommodation which the patient contributes when using the device. These formulas specify the increase in size of the retinal image relative to its size when the same object is viewed without the magnifier from a distance of 40 cm. Determination of the size of print which the patient can read at 40 cm. gives therefore a direct measure of the magnification required to read ordinary newspapers, magazines, and books.

The Johns Hopkins Hospital (5).

Appendix 1

Derivation of formulas for M_{40}

In the upper diagram of Figure 2, S represents an object located at a distance of 40 cm. from F, the anterior principal focus of the eye. In the lower diagram of the same figure, the same object S is viewed through a convex lens of D diopters, located at a distance h from F. It is assumed that the object-to-lens distance, u, is less than the principal focal length of the lens so that an enlarged virtual image s' is formed at a distance u' from the lens.

In dealing with relatively large increases in size of the retinal image, we can ignore the small change in the location of F which occurs with increase of the accommodative state of the eye, so that for a fixed anterior focal distance

$$M_{40} = w'/w$$

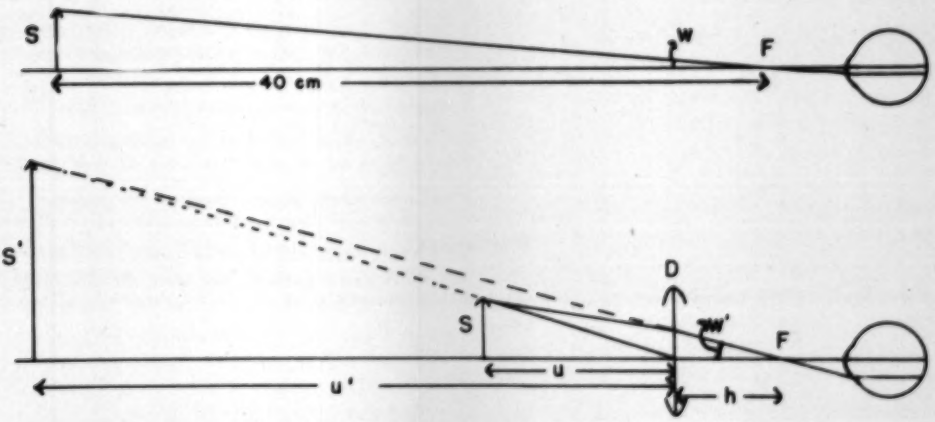

Fig. 2 (Sloan and Habel). To show angular magnification relative to a reference distance of 40 cm. (w) is the visual angle subtended by the object when it is viewed without the magnifier: (w'), the visual angle subtended by its virtual image when the magnifier is used.

If the distances u, u′, and h are taken as positive, and if all distances are in meters,

$$w = s/0.4 = 2.5s$$

$$w' = \frac{s'}{u'+h}$$

$$\frac{1}{u'} = \frac{1}{u} - D$$

The accommodation A in diopters (measured from F) which the emmetropic eye must exert to obtain a clearly focused image of s′ is equal to

$$\frac{1}{u'+h}$$

Since

$$A = \frac{1}{u'+h} \qquad u' = \frac{1-hA}{A}$$

$$M_{40} = \frac{w'}{w} = \frac{s'}{s} \times \frac{A}{2.5} = \frac{u'}{u} \times \frac{A}{2.5}$$

$$\frac{1}{u} = D + \frac{1}{u'} = D + \frac{A}{1-hA} = \frac{D-hAD+A}{1-hA}$$

$$\frac{u'}{u} = \frac{1-hA}{A} \times \frac{D-hAD+A}{1-hA} = \frac{D+A(1-hD)}{A}$$

$$M_{40} = \frac{D+A-hAD}{2.5} \qquad \text{(a)}$$

Formula (a) which specifies M_{40} in terms of D, A, and h is useful for focusable magnifiers in which, for fixed values of D and h, A varies with the object-to-lens distance u.

When u is fixed for a given magnifier, and h and A are the dependent variables, a formula is needed which specifies M_{40} in terms of D, A, and u. This may be derived as follows:

$$M_{40} = \frac{u'}{u} \times \frac{A}{2.5}$$

$$\frac{1}{u'} = \frac{1}{u} - D = \frac{1-Du}{u}$$

$$\frac{u'}{u} = \frac{1}{1-Du}$$

$$M_{40} = \frac{1}{1-Du} \times \frac{A}{2.5} \qquad \text{(b)}$$

The relationship between A and h for fixed values of D and u may be derived as follows:

$$u'+h = \frac{1}{A} = \frac{u}{1-Du} + h \qquad \text{(c)}$$

Appendix 2

TABLE 1

VALUES OF M_{40} FOR REPRESENTATIVE MAGNIFIERS

Magnifier and Source*	M_{40} for A equal to 0.0 diopters	2.5 diopters	5.0 diopters
Plano-convex, AFB	—	1.7	3.4
3d add	1.2	2.2	3.2
4d add	1.6	2.6	3.6
5d add	2.0	3.0	4.0
Projection Magnifier, 3×, AO	—	3.0	6.0
Plasta Cataract, AFB	—	3.1	6.2
6d add	2.4	3.4	4.6
8d add	3.2	4.2	5.2
Magnifocuser, AFB	4.0	5.0	6.0
10d add	4.0	5.0	6.0
Projection Magnifier, 5×, AO	—	5.0	10.0
Policoff 12d	4.8	5.8	6.8
Linen Tester, B&L	8.0	(8.0)†	(8.0)†
Policoff, 20d	8.0	9.0	10.0
Adisco, AFB	8.4	(8.4)†	(8.4)†
Policoff, 28d	11.2	12.2	13.2
Tripod Magnifier, AFB	11.2	12.2	13.2
Flaw Finder, AFB	11.4	12.25	13.1
Megascope 12×, AFB	—	12.0	24.0
Tripod Magnifier, B&L	12.3	13.3	14.3
Policoff, 40d	16.0	17.0	18.0
Megascope 25×, AFB	—	25.0	50.0

* The designation AFB indicates that the magnifier can be obtained from the American Foundation for the Blind, 15 West 16th Street, New York 11, New York. The Policoff lenses are manufactured by Dr. Wm. M. Policoff, Wilkes-Barre, Pennsylvania. The designations, AO and B are used to designate respectively magnifiers of the American Optical Co. and Bausch & Lomb Optical Co.

† Since these magnifiers are prefocused for infinity, the observer cannot increase the size of the retinal image by adding his own power of accommodation to the dioptric power of the magnifier.

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DISTURBANCES OF OCULAR MOTILITY IN RELATION TO SUPRANUCLEAR LESIONS*

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I. INTRODUCTION

There should be no need to emphasize the importance of eye signs in the diagnosis of intracranial lesions. Although the treatment of these lesions lies primarily within the province of the neurologist or neurosurgeon, their recognition and diagnosis often devolves upon the ophthalmologist because the presenting complaint is so frequently ocular in nature. In addition, the ophthalmologist should be of immeasurable assistance in the more precise localization and identification of such lesions because of his specialized knowledge of diseases not only of the eye but those related to it. He must realize his limitations as regards the treatment of these disorders and should be prepared, when the situation warrants, to avail himself of adequate consultation. It must be remembered that, by definition, these lesions are located within the cranial vault and are, strictly speaking, outside the realm of ophthalmology as far as treatment is concerned. Whether such treatment is medical or surgical in nature is beside the point; the case represents more than strictly an eye problem.

This discussion is limited to those disorders of the supranuclear pathways which produce disturbances in ocular motility. And, more precisely, it is limited to acquired pathologic disorders as opposed to, for example, concomitant strabismus. It must be remembered that these disturbances may not represent the only eye findings in cases of intracranial lesions. The appearance of the fundus and the analysis of the visual fields are also pertinent in most cases, but are outside the scope of the discussion. Time does not permit the consideration of disorders resulting from damage limited to the oculomotor nuclei themselves, or the infranuclear pathways to the ocular muscles.

The lesions under consideration are thus only those which involve the cortex and the cerebral pathways related to the nuclei of the motor nerves supplying the eye; namely, the III, IV, and VI cranial nerves. It was Gowers, in his monumental work entitled *Manual of Diseases of the Nervous System*, published in 1886, who first pointed out the differences in manifestations between these supranuclear lesions and lesions of the nuclei themselves, or the infranuclear peripheral nerve trunks.

Basically, infranuclear lesions produce palsies or spasms which can be analyzed in terms of the individual extraocular muscles themselves. Supranuclear lesions, on the other hand, cause interference with overall movement of the eyes rather than with movements of individual muscles. These overall movements include conjugate lateral, upward, and downward gaze (termed "versions" by the ophthalmologists), as well as the disjunctive movements of convergence and divergence. In most instances parallelism of the visual axes is maintained, except in disturbances of convergence and divergence and in the syndrome of internuclear ophthalmoplegia. For this reason, diplopia is less frequently a complaint in supranuclear disorders than in infranuclear diseases.

In the case of supranuclear lesions the action of the individual extraocular muscles remains intact. For example, in paralysis of conjugate lateral gaze, voluntary adduction of one or the other eye is not possible, and yet bilateral adduction, as demonstrated by convergence, may be easily accomplished. By the same token, voluntary upward gaze

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may be impossible, but Bell's phenomenon remains intact, proving that the infranuclear pathways are undisturbed.

It must be kept in mind that the lesions with which we are dealing may be either irritative or destructive in nature. On the one hand, overstimulation results, and there is an overaction of the muscle groups involved; on the other hand there is paralysis and underaction. It is not uncommon for a lesion originally irritative in nature eventually to become destructive, thereby producing a reversal of the original manifestations. It must also be remembered that in no case are abnormal activities in one group of structures isolated in their manifestations, and we should expect abnormal activities to take place in another group of undamaged related structures. This is due to the modification of whatever controls and inhibitions are normally exercised upon them by the organism as a whole. This concept, which was first stressed by Hughlings Jackson, is of considerable importance in dealing with any intracranial lesion.

The central connections of the various oculomotor nuclei are extremely complex in nature and the exact pathways have by no means been identified. It is convenient, however, to discuss the subject under four headings, involving four separate, although related, types of control of ocular motility. The mechanisms involved are:

1. The frontal mechanism, which is concerned with the control of voluntary conjugate movements.

2. The disjunctive mechanism which controls convergence and divergence. This is thought to be influenced by both the frontal and occipital mechanisms.

3. The occipital mechanism, which controls involuntary reflex movements. Examples are the following and fixation reflexes.

4. The extrapyramidal mechanism, consisting of the vestibular system and the striatal system. These control respectively the righting reflex and the tonus and co-ordination of the extraocular muscles.

II. Anatomy

Before considering the disturbances produced by intracranial lesions involving the four mechanisms just mentioned, it would be well to review briefly the anatomy of the pathways involved.

1. *Voluntary conjugate movement* of the eyes is produced by stimulation of area 8 of Brodmann, which lies in the caudal portion of the second frontal convolution. Stimulation of this oculogyric area on one side results in conjugate deviation of the eyes to the opposite side. Leaving area 8, the efferent fibers pass downward through the corona radiata to the cerebral peduncle near the genu of the internal capsule, in close apposition to the supranuclear fibers for the facial muscle. They then course through the basal ganglia and terminate in the rostral portion of the pons. Complete decussation of the tract occurs in the midbrain, probably near the posterior commissure, and prior to decussation of the facial fibers. In the midbrain the fibers for conjugate vertical movement leave the tract and course directly along the brachium to the superior colliculi. Those for lateral movement run caudally to the pons in the region of the VI-nerve nuclei. Here they are integrated with impulses of visual, auditory, and proprioceptive origin. This integration is logically accomplished through intermediary centers, prior to stimulation of the individual motor nuclei themselves. There is considerable evidence in favor of the existence of centers for conjugate vertical and lateral gaze, although their existence is not universally accepted nor has it been proven. The center for conjugate lateral gaze is probably near the VI-nerve nucleus, and that for vertical gaze is most likely the superior colliculus.

2. *Affections involving disjunctive movements* are not nearly as common as those involving conjugate gaze. Convergence has been produced by bilateral stimulation of the frontal and occipital lobes, but the observations have been too inexact to permit of any precise localization of function with-

in the cortices. The subcortical center for convergence is most probably the nucleus of Perlia, which is an unpaired group of motor cells lying between the III-nerve nuclei. This nucleus develops phylogenetically along with the function of convergence. It receives impulses from the frontal oculogyric center through the corticonuclear tract by way of the superior colliculi. It also receives reflex stimuli from the occipital cortex by way of the occipitomesencephalic tract. Since there is frequently dissociation of convergence from lateral conjugate gaze, it is probable that the convergence pathways do not travel by way of the pons.

The existence of a divergence center is somewhat debatable, and evidence for it is only presumptive. The concept of a divergence center depends almost entirely upon the occurrence of a syndrome described first by von Graefe, and subsequently by Priestley Smith, Parinaud, and Duane. Here, convergent strabismus suddenly develops along with uncrossed diplopia for distance, but without any limitation in function of either lateral rectus muscle. The location of the divergence center, if present, is not well-established, but autopsy records suggest that it is located in the brain stem close to the level of the VI-nerve nucleus.

3. *In contradistinction to the voluntary movements* of the eyes which are originated by the frontal cortex, the occipital cortex is primarily concerned with the more primitive reflex movements controlling the eyes. These movements concern primarily fixation and fusion, as well as involuntary convergence and accommodation. These reflexes are largely involuntary, develop later in life, and are, in general, subservient to the movements initiated by the frontal cortex. They have been termed the "Psycho-optical reflexes" by Bielschowski. The occipital regions involved are about the calcarine fissure (area 17), the parastriate region (area 18), and the peristriate region (area 19).

Stimulation of one lobe of the occipital cortex results in conjugate deviation of the eyes to the opposite side, although there is not the precise localization which is found in area 8. In monkeys, the lowest stimulation threshold is in area 17; areas 18 and 19 require respectively larger potentials. Even though there is no direct connection between the two frontal oculogyric centers, each is connected to the homolateral occipital lobe by means of superficial association fibers. It is probable that a tonic depressant effect is exerted by the occipital centers upon the frontal centers, although the frontal centers appear to be dominant. In addition, unlike the frontal centers, the occipital centers are connected through the corpus callosum and appear to exert a mutual inhibitory effect. Direct connections between the occipital cortex and the pontine centers must also be present because destruction of the frontal centers does not result in the abolishment of conjugate movements on occipital stimulation. The efferent fibers form a well-defined fasciculus and course parallel with and medial to the visual radiation. They pass through the posterior end of the internal capsule and terminate in the rostral portion of the pons. Fibers have been traced to the thalamus, superior colliculi, and other portions of the mesencephalon. It is probable that, on the basis of evidence available, the occipital motor fibers are relayed through the superior colliculi while the frontal fibers are relayed through the basal ganglia.

4. *The extrapyramidal control of ocular motility* rests within two systems. The octavus, or VIII-nerve system, is concerned with postural functions and the striatal system is concerned with the maintenance of muscle tonus and rhythm. The octavus system is in turn composed of two divisions, the cochlear and the labyrinthine, the activities of which are co-ordinated by the cerebellum. Since the cochlear, or acoustic, division plays little part in the control of ocular movements, we will limit our discussion to the labyrinthine pathways of the octavus system.

The actual pathways for labyrinthine

stimuli are not well understood but are probably through the intermediate pontine centers governing conjugate deviations, by way of the vestibular nuclei. Each labyrinth sends tonic impulses to the vestibular nucleus of the same side which in turn influences the motor nuclei of the opposite side, causing movements of the eyes to that side. The stimuli may be either from the semicircular canals (opticokinetic) or from the otolith apparatus (opticostatic). Impulses from the left labyrinth, therefore, cause conjugate deviation to the right. By the same token a destructive lesion of one labyrinth will cause conjugate deviation to the injured side, or a nystagmus with the quick component directed to the sound side. It should be remembered that the quick, or recovery, component in vestibular nystagmus is not a function of the vestibular apparatus per se, and may be absent.

The striatal system, which is phylogenetically the oldest part of the mechanism, occupies the region between the diencephalon and cerebral cortex. It is composed of the corpus striatum, the thalamus, the hypothalamic nucleus of Luys, the substantia nigra, and the red nucleus. There are elaborate connections between the cortex and the striate region, and it is probable that it plays a subordinate reinforcing role to the cortex and does not act autonomously. The rhythm, precision, and co-ordination of normal ocular movements are controlled here as well as the tonicity of the extraocular muscles. As previously mentioned, the subcortical pathways from the frontal oculogyric centers are probably relayed through these basal ganglia.

In connection with the activities of the octavus system, a brief discussion of the part played by the cerebellum is in order. This organ is purely motor in nature and co-ordinates the activities of the motor mechanism in general. It arises as an outpouching of the vestibular nuclei, and consists of a central unpaired portion, the vermis, and the paired lateral lobes. The older portion, the vermis, subserves equilibration and posture, while the lateral lobes, or neocerebellum, subserve motor co-ordination. Fibers from the cortex of the vermis are projected to the underlying nuclei in the roof of the fourth ventricle and thence to the VI-nerve nuclei and medial longitudinal fasciculus by way of the superior cerebellar peduncle. Stimulation of the vermis and roof nuclei produces vertical conjugate movement of the eyes. On the other hand, stimulation of the lateral lobes, or of the intracerebellar dentate nuclei, results in deviation of the eyes to the homolateral side. Removal of large portions of the lateral hemispheres results in homolateral hypotonia, weakness, and dysmetria of the skeletal muscles. In general, then, the vermis is generally associated with the maintenance of conjugate vertical gaze, and the lateral lobes with the maintenance of conjugate lateral gaze to the same side.

A discussion of the anatomy of the supranuclear pathways would not be complete without a description of the medial longitudinal fasciculus, or, as it is sometimes called, the posterior longitudinal bundle. This tract extends on either side of the midline from the thalamus anteriorly to the anterior horn cells of the spinal cord posteriorly and is especially well-developed in the region between the nuclei of the oculomotor and vestibular nerves. It is made up in a large part by fibers from the vestibular nuclei. It serves to co-ordinate the motor nerves of each eye with those of the other, as well as with the vestibular nuclei, and relays impulses from the pontine centers for conjugate lateral gaze. It should be remembered that impulses for conjugate vertical gaze arrive by way of the superior colliculi and are not mediated by this tract. The lateral rectus muscle, being directly innervated by the lateral gaze centers, also remains uninvolved with the medial longitudinal fasciculus. The centers for lateral gaze are found on the homolateral portion of the tract inasmuch as the cortical fibers have already

decussated before reaching it. Fibers destined for the opposite medial rectus probably cross the midline posteriorly near the lateral gaze centers and course medially in the fasciculus of the opposite side. Thus destructive lesions located on one or the other side of the midline in the medial longitudinal fasciculus will produce paresis of the homolateral medial rectus muscle. This is the basis of the syndrome of internuclear ophthalmoplegia, which we shall discuss later.

III. Types of Motor Anomalies

In discussing the types of motor anomalies produced by supranuclear lesions, it is convenient to classify these under the same headings used to describe the anatomic pathways involved.

1. Lesions of the Central Mechanism for Voluntary Control

Destructive lesions of the central mechanism for voluntary ocular control produce conjugate, or gaze, palsies to the side opposite the lesion. The patient is unable to move his eyes in a given direction upon command, and efforts to do so are unavailing and may be associated with considerable distress. Because of the loss of the normal inhibitory influences, as expressed by Sherrington's law, there is an increased tonicity of the antagonistic muscles, which results in a deviation of the eyes to the side of the lesion. This may be compensated for by turning the head slightly toward the side opposite the lesion. Jerky nystagmus toward the affected field of gaze may result from voluntary efforts on the part of the patient to control his lateral movement. Diplopia is not a common complaint inasmuch as both eyes have been equally affected.

In gaze palsies the fixation reflex is not only unaffected but is, in fact, intensified. The patient is able to follow a slowly moving object in the paralyzed field of gaze in spite of the lack of voluntary control, providing there are no large gaps in the line of attention. Gaze may become so anchored to the object of fixation (spasmodic fixation) that posture of the eyes can only be changed by covering them. In this case the exaggerated tone of the ocular muscles is due to the effect of retinal impulses acting upon a cortical mechanism no longer under inhibitory control.

Lesions of the frontal centers usually produce transient disturbances, probably due to the assumption of control by the undamaged hemisphere. For this reason, also, disturbances are most evident with acute lesions and are absent with chronic lesions. In the case of acute lesions, such as those due to vascular accidents or trauma, a conjugate deviation to the side of the lesion may only be present during the period of unconsciousness. When the patient awakens there may be either no manifest disturbance or only a slight nystagmus with the quick component to the opposite side. Should the patient again lose consciousness, or be anesthetized, the deviation may reappear. Other signs usually seen in relation to frontal lobe lesions are hemiplegia and head turning toward the side of the lesion.

Impulses arising in the labyrinths and neck muscles are not interfered with in lesions of the frontal voluntary gaze centers. If, for example, there is paresis of voluntary right lateral gaze, and should the head be suddenly twisted to the left, the eyes will rotate to the right, into the paralyzed field. Upon the cessation of this transient impulse the eyes will then slowly slide backward to their original position. This is the so-called "Doll's head phenomenon." A somewhat similar reaction may occur because of the fixation reflex, but the fact that it will be observed in blind eyes indicates that two separate mechanisms are involved. Its dependence upon the postural mechanism is illustrated by its occurrence in patients in whom vestibular reactions are otherwise entirely lacking.

Gaze palsies can also be produced by cortical lesions other than frontal in location. Be-

cause of the complex associations of function of the frontal with the occipital, parietal, and temporal lobes, lesions of these latter areas may produce the same results. Insofar as cortical lesions are concerned, therefore, gaze palsies have primarily a lateralizing rather than a localizing value in diagnosis. Lesions of the cortices are not, however, the most common cause of palsies of voluntary gaze.

Lesions involving the subcortical pathways may also result in gaze palsies, which are more often permanent in nature. This is probably because, in the case of cortical lesions, there is eventual assumption of control by the opposite hemisphere. Either lateral or vertical conjugate movements may be affected in subcortical affections and, in contradistinction to those involving the cortex, bilateral destructive lesions here may permit the maintenance of vertical movement when no horizontal movement is possible. Subcortical lesions can usually be identified by the presence of more prominent associated signs, particularly hemiplegia, and, in the case of pontine lesions, by associated peripheral nerve involvement which results in diplopia. Bilateral involvement of conjugate gaze is also more frequently seen in pontine lesions because of the near midline situation in the pontine centers. Paresthesias are common in the pontine syndrome because of involvement of the lemnisci. The hemiplegia, which may be present due to the involvement of the pyramidal tracts, is to the opposite side of the conjugate gaze defect in pontine lesions whereas it is to the same side in cortical lesions. The most common cause of pontine lesions are vascular accidents, particularly thrombosis of the basilar artery, although tumors, multiple sclerosis, and encephalitis must be considered. Some epidemics of encephalitis have been reported to demonstrate preferential involvement of lateral conjugate movements.

If a destructive lesion is situated above the decussation of the corticonuclear tract in the midbrain, paralysis of conjugate movement to the opposite side will result, similar to that occurring from cortical lesions. A lesion at the decussation will produce paralysis of conjugate lateral gaze to both sides, and a lesion below the decussation will produce paralysis to the homolateral side. Since the center for convergence lies at a higher level than that of lateral gaze (being reached through the superior colliculus), a lesion at a higher level may result in abolition of convergence along with lateral gaze, while a lesion situated lower in the tract may allow retention of convergence.

Lesions due to irritative disorders. So far, we have considered only destructive types of lesions. However, conjugate deviation of a spasmodic nature on the basis of irritative disorders also occurs, although less frequently. As previously stated, a lesion which may be at first irritative may later become destructive, in which case a spastic deviation to one side is replaced at a later stage by a paretic deviation to the opposite side. In contradistinction to paretic lesions, conjugate spasm is usually cortical rather than subcortical, and usually involves adjacent areas of the cortex as well, resulting in head turning and contraction of the limbs.

Irritative lesions involving the frontal oculogyric centers are most commonly seen in cases of Jacksonian epilepsy, in which there is a clonic turning of the eyes and head toward the side opposite the lesion followed by a generalized convulsion. With lesions in area 8 the turning of the eyes precedes that of the head, while with lesions in areas 6 and 9 the order is reversed. Unless the spasm is excessive, voluntary movement to the opposite side can still be accomplished.

Subcortical lesions producing conjugate spasmodic deviation are less common, although their symptoms may be almost the same. Occasionally these may result in the head being turned one way and the eyes opposite, indicating a lesion destroying one,

and irritating the other, of the closely related occulogyric and cephalogyric tracts. Conjugate spasmodic vertical deviations are essentially extrapyramidal in origin and are therefore rarely seen in true convulsive disorders. Their most common occurrence is in the so-called oculogyric crises which we will discuss subsequently.

Disorders of conjugate vertical gaze are rarely, if ever, produced by cortical lesions, presumably because of the bilateral representation and the assumption of control by the opposite lobe. If a vertical component is present at all, it is associated with some disorder of lateral gaze. However, since vertical and horizontal functions are separately represented in the brain stem, vertical gaze disturbances are particularly valuable from the standpoint of localization. Most commonly they indicate a lesion of the subthalamic region in the vicinity of the posterior commissure. Most frequently, upward gaze alone is involved; less frequently both upward and downward gaze, and least frequently downward gaze alone.

Although the precise localization of lesions which may affect vertical gaze is indefinite, it would appear on clinical grounds that the center for upward movement is at a higher level than that of downward movement, inasmuch as lesions extending downward into the midbrain from the third ventricle produce first a paralysis of upward gaze and later a paralysis of downward gaze. If the lesion is in close association with the superior colliculus, loss of conjugate lateral gaze, as well as convergence, may occur along with loss of vertical gaze.

There may also be inequality of the vertical paralysis so that vertical diplopia results. The paralysis of upward gaze, which is seen in Parinaud's syndrome, is most frequently due to a tumor of the pineal gland but can also result from lesions within the superior colliculi or thalamus, as well as from neurosyphilis, encephalitis, multiple sclerosis, or vascular accidents. Paralysis of vertical gaze is almost always accompanied by pupillary disturbances and weakness of convergence. Vertical gaze palsies have also been reported in association with lesions of the fourth ventricle and cerebellum, probably the vermis, and differ from those resulting from lesions in the region of the colliculi in that normal pupillary reactions are maintained. As in disorders of lateral gaze, if the following reflex is intact, the patient may be able to follow a slowly moving object into the paralyzed field of gaze.

In summary, the various types of conjugate palsies, or gaze palsies, can be classified as follows:

a. Loss of conjugate movement of both eyes to one side with retention of all reflex activities, in association with conjugate deviation of the eyes to the opposite side. If the deviation is temporary, the lesion is probably cortical, and if permanent, probably subcortical.

b. Bilateral loss of conjugate lateral movement due to a lesion in the neighborhood of the posterior commissure.

c. Loss of conjugate lateral movement associated with loss of conjugate vertical movement, produced by a lesion in the vicinity of the superior colliculus, or due to bilateral cortical involvement.

d. Loss of conjugate movement to one side, associated with loss of convergence, due to extensive lesions in the pons involving the centers for conjugate gaze, as well as for convergence.

2. Disjunctive deviations

Disjunctive deviations are due to affections of the centers for convergence and divergence and are much rarer than conjugate deviations. Functional deficiencies of convergence are quite common, but organic paralysis is relatively rare. It is probable that convergence is a bilateral cortical function and that it is partially voluntary and partially involuntary. Normally it is closely associated with miosis and accommodation, the triad being known as the near reflex, but these may become dissociated in certain

cases. A very small lesion in the midbrain may produce a pure convergence palsy if only the nucleus of Perlia is affected, but more often the pupillary action of miosis is lost as well, resulting in the so-called "reverse Argyll Robertson pupil." Frequently accommodation is also paralyzed. In Parinaud's syndrome, convergence paralysis is characteristically associated with paralysis of accommodation and vertical gaze.

The syndrome of convergence palsy is characterized by a failure of convergence and consequent diplopia for near objects, together with the absence of limitation of ductions or versions. Care must be taken to differentiate true convergence palsy from hysteria or lack of interest. Suggestive of a true paralysis are the history of sudden onset of diplopia for near, the constancy of findings on successive trials, or the presence of a known intracranial lesion. Characteristically it occurs with rather discrete lesions in the vicinity of the superior colliculi in association with vertical gaze palsies, but it can also be caused by more diffuse disorders such as encephalitis, multiple sclerosis, and Parkinson's disease.

Isolated convergence spasm is extremely rare but, in association with spasm of accommodation and miosis, it is somewhat more common. This is the so-called "spasm of the near reflex" which is seen in irritative conditions as well as in hysteria.

We have already mentioned the syndrome of von Graefe, Priestley Smith, and Duane which supports the existence of a divergence center. Here, accommodation is unaffected, as is convergence, and the diplopia tends to increase on downward gaze and decrease on upward gaze. It can be differentiated from bilateral VI-nerve palsies by the presence of normal ductions, and from spasm of the near reflex by the absence of accommodative and pupillary dysfunction. The diagnosis should be made with caution inasmuch as a functional divergence insufficiency, or latent esotropia, may produce much the same pattern. The etiology of divergence palsy is generally similar to that of convergence paralysis. It is seen in encephalitis, tabes, multiple sclerosis, following trauma, and with tumors and cysts of the cerebellum. Alternating palsies of divergence and convergence have been noted in Wernicke's disease, a fact which would suggest that the convergence and divergence centers, or tracts, are closely associated in at least one portion of the brain.

In connection with disjunctive deviations we should just briefly mention the syndrome of dissociated vertical divergence. This was termed "anaphoria" by Stevens, "alternating hyperphoria" by Duane and Bielschowsky, and "occlusion hypertropia" by Verhoeff. At the present time it is commonly referred to as alternating sursumduction, abbreviated A.S.D. In this condition each eye, when covered, will move upward while the fellow eye is fixating. In rare instances, the eyes move downward under cover. This disturbance is primarily innervational and is not due to any acquired lesion of the supranuclear pathways.

3. Primitive reflex movements

The more primitive of reflex movements controlling ocular motility are associated with the occipital cortex and include the fixation and fusion reflexes and the blink reflex, as well as involuntary convergence and accommodation. They do not include fixation movements initiated by attention or interest and they cannot be produced or stopped voluntarily. In general, we can say that an oculomotor center exists in each occipital lobe which is dominated by the frontal centers, and which is concerned with the ocular movements required by the psycho-optical reflexes.

Obviously, diseases of the macula or the presence of central scotomas will result in the abolishment of these reflexes, and the normal fixation movements are replaced by a series of jumps, so-called cogwheel move-

ments. This resembles a primitive stage in the development of the fixation reflex. This same disturbance occurs in diseases involving the optic pathways whether total, as in the case of blindness, or partial, as in the case of hemianopsias. Primary disorders involving the occipital cortex itself, in which its sensitivity is lowered or disoriented, will also result in a disturbance of the psychooptical reflexes.

In bilateral occipital lesions the ability to maintain fixation is almost completely lost. The eyes oscillate about the object of fixation and cannot follow moving objects. Voluntary movement is possible but is not supported and maintained by the fixation reflex. If the lesion is unilateral, the symptoms are less pronounced but difficulty in maintaining fixation may be considerable.

Defects in opticokinetic nystagmus also occur toward the side of the occipital lesion, but of primary importance in the differential diagnosis of occipital lesions are the associated visual field defects, which are, in fact, usually more conspicuous than are the motor disturbances. Disturbances in motility, per se, can properly be studied only in those rare instances in which only the efferent fibers are involved and the closely associated optic radiations are spared.

In the syndrome of pseudo-ophthalmoplegia, both command movements and following movements are affected, although usually unequally. This is indicative of a diffuse process involving the corticofugal fibers from both the frontal and occipital cortices and represents little in the way of localizing value.

It must be remembered that inability to fix may be due to defective vision, central scotomas, nystagmus, or muscular palsy, and that none of these are present in cases of true fixation palsy.

4. Extrapyramidal control

As previously mentioned ***the extrapyramidal control of ocular movement*** rests with the octavus system, which subserves a postural and co-ordinating function, and the striatal system, which is concerned with the maintenance of muscle tonus and rhythm. We have already seen that impulses from one labyrinth cause conjugate deviation to the opposite side and that, therefore, a destruction of one labyrinth will produce conjugate deviation to the injured side. Excitation of the labyrinth by caloric, galvanic, or rotary stimuli can therefore be used in the differential diagnosis of conjugate lateral deviations, as follows:

a. If the labyrinth or its central connections have been destroyed, no nystagmus or head turning reflex will be elicited.

b. If the lesion is peripheral or infranuclear, only the unaffected eye will move.

c. In the cases of supranuclear or cortical palsies there will be slow conjugate deviation toward the stimulated side since the rapid restitutory phase of nystagmus is absent due to lack of cortical control.

Perhaps the most important eye findings in the case of vestibular disturbances are those concerned with torsional conjugate movement. Apparently, these movements have no representation in the frontal lobe and little in the occipital lobe, but are almost exclusively controlled by the labyrinths. This torsion tends to maintain the original orientation of the eyes in relation to the environment, but only to a partial degree. The stimulus for this comes partially from the neck muscle, but chiefly from the labyrinths. Because of this, rotary nystagmus is prima facie evidence for vestibular involvement and is especially frequent in lesions of the vestibular nuclei.

5. Lesions of the striatal system

Lesions of the striatal system which affect control of extraocular movements are characterized by the following:

a. *The appearance of abnormal involuntary movements.* These are probably due to a disorganization of the activity of the cor-

pus striatum, which leads to a condition resembling ataxia. They are characteristically seen in the so-called oculogyric crises of postencephalitic Parkinsonism, as well as in chorea, athetosis, and hepatolenticular degeneration.

b. *Hesitancy of voluntary movement.* Destruction of striatal nerve tissue may result in a loss of the normal reinforcement and strengthening of movements originating in the cortices. This hesitancy has been observed most frequently in destructive lesions of the red nucleus.

c. *Tremor.* This is probably due to the loss of the regulating influence of the globus pallidus and substantia nigra. The tremor is one of active posture as opposed to the tremor of cerebellar disease which is one of active movement.

d. *Changes in muscular tonicity.* Lesions of the striatal system may either produce an increase in muscle tone, as in the case of Parkinsonism, or hypotonia, as in athetosis and chorea.

Cerebellar lesions most commonly produce horizontal nystagmus with the quick component toward the side of the lesion, the oscillations becoming greater when the eyes are deviated toward the side of the lesion. For this reason head turning to the side of the lesion is a common finding in order that the eyes may find a position of rest. The manifestations of cerebellar disease have been interpreted as an inability to maintain conjugate gaze to the side of the lesion and only rarely does complete paralysis occur. Tumors, particularly astrocytomas and medulloblastomas, as well as abscesses, are among the more common cerebellar lesions.

So far we have considered only those disorders which are characterized by a more or less equal affection of each eye, but there are certain conditions, known as dissociated deviations, in which the eyes are affected unequally or in varying manner, such as one might expect in the case of nuclear or infranuclear disorders.

The most common type of dissociated deviation is seen in the syndrome of internuclear ophthalmoplegia. This syndrome is produced by lesions of the medial longitudinal fasciculus, resulting in the unharmonious co-ordination of the various nuclei involved in the production of conjugate gaze. If the lesion is in the anterior portion of the tract the lateral rectus muscles will function normally, but one or both medial rectus muscles may be paralyzed for conjugate movements. Convergence is normally maintained but may be decreased. In lesions involving the posterior portion of the tract both internal rectus muscles function normally in convergence and lateral gaze, but the lateral rectus on the side of the lesion is weakened for conjugate movements.

Dissociated vertical palsy is a rare condition and is due to a localized lesion close to the nuclei below the point where the corticofugal pathway for elevation bifurcates into the branches which go to both III-nerve nuclei.

One other dissociated deviation of some importance is "skew deviation," also known as the Hertwig-Magendie syndrome. This may be encountered in acute cerebellar lesions such as trauma, abscesses, and neoplasms. The eyes are spasmodically deviated in different directions, the eye on the affected side looking down and inward while the opposite eye looks upward and outward.

IV. Conclusion

An attempt has been made to describe briefly the most common types of disorders of ocular motility related to lesions in the various portions of the supranuclear pathways. Numerous isolated syndromes have been omitted in favor of a more generalized approach to the matter. It is hoped that this will enable the ophthalmologist to interpret more quickly and accurately the manifestations of central nervous system disease which he may encounter.

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KERATOTOMY IN MOOREN'S ULCER OF THE CORNEA*

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Mooren's ulcer or chronic serpiginous ulcer is a slowly progressive ulcer of obscure etiology which begins at the corneal periphery and undermines the border as it progresses about the circumference. It usually occurs in elderly people. It is accompanied by extraordinary pain and frequently results in complete corneal opacification and often loss of the eye. The problem it presents for present-day ophthalmologists is almost as baffling as it was when it was first described 90 years ago.

Despite the efforts of over 100 known authors on the subject, our knowledge of the etiology remains obscure and our treatment continues to be uncertain and unpredictable. Considerable information has been made available through published reports so that by the accumulation of data the unknown factors are perhaps less numerous than formerly. Linn[1] has reported on an analysis of 53 cases of chronic serpiginous ulcer which have been cured. From his data, it is concluded that beta radiation, delimiting keratotomy, and a conjunctival-flap operation are the most effective therapeutic measures.

Numerous attempts at bacteriologic studies have been unrewarding. In certain instances inoculations have been negative. Thygeson[2] studied six cases exhaustively for a causal microbe but without success. Chemotherapy and antibiotic therapy have been singularly ineffective in halting the progress of Mooren's ulcer.

Metabolic disorders and malnutrition have been considered. Several authors, Junius[7] and Triebenstein,[8] described a trophic disturbance due to fifth-nerve changes as the primary cause. It may also be postulated that a diminished nutritional supply secondary to arteriosclerosis at the limbus could be a factor. Bacterial invasion is probably secondary. Cogan[9] has reported on certain corneoscleral lesions which may be a characteristic manifestation of periarteritis nodosa. The corneal lesion consists of a furrowlike ulceration in the paralimbal region which in the severe cases has all the features of a classical Mooren's ulcer. The pathologic basis for these lesions appears to be an occlusive vasculitis.

Histologic examination of an eye with Mooren's ulcer usually shows no particular characteristics. It shows an essentially necrotizing process surrounded by a round-cell infiltration. The overhanging margin of the cornea is necrotic and the floor of the ulcer is covered with granulation tissue. As the active part of the ulcer encroaches on the center, the periphery of the cornea heals with a thin layer of highly vascularized scar tissue. An opaque scar indicative of healing gradually replaces the transparent cornea.

Thygeson attained partial success by the

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use of delimiting keratotomy combined with repeated paracenteses, according to the technique of Gifford. The ulcers were apparently arrested in three cases while in the remaining three cases they progressed centrally and extended over the keratotomy incisions.

A mucous membrane graft from a patient's lip has been used in several instances and has been credited with initiating a definite cure.[3]

Although cauterization has apparently cured more cases than any other method, it has also been reported ineffective for the greatest number of patients with Mooren's ulcer.

More recently, Paton[4] has reported on the excellent results from corneal grafting in the early cases of chronic serpiginous ulcer. A lamellar ring-shaped graft is sutured directly into the prepared defective area.

Clinical characteristics

Mooren's ulcer is a relatively rare disease. In the available literature it was unusual for one ophthalmologist to have reported more than three cases of his own even after working for many years.

Penido Burnier[5] reported four cases from a total of 102,000 eye patients.

In over 25 percent of cases it is bilateral but several years may elapse between involvement of each eye.[6] It characteristically occurs in elderly people but case reports include all ages.

The disease begins with a gray patch of infiltration at the corneal margins approximately a mm. from the limbus. The ulcer spreads slowly along the margin and gradually in toward the center of the cornea. After the ulcer has been present for two weeks or longer, a dehiscence or furrow becomes apparent along with an overhanging margin with its base on the corneal side. The ulcer penetrates about half way through the cornea and never appears to go deeper. Perforation is rare. Healing takes place very slowly from the periphery with the development of new vessels and scar tissue. In front of the advancing border there is a gray infiltration and beyond this point the cornea remains completely clear.

Symptoms of pain and photophobia are very severe in spite of the mild tissue response to inflammation. The eye pain is not controlled by the use of local anesthetics. In addition to the local eye discomfort, the pain seems to involve most of the area supplied by the first division of the fifth nerve. Even heavy sedation may give but temporary relief and the increasing exhaustion of the patient becomes as much of a problem as the eye. Unfortunately, in too many cases, despite all treatment available, the pain and ulcer continue unabated and the prognosis for future vision becomes poor.

Case report

A case involving both eyes was successfully treated by delimiting keratotomy after a conjunctival flap failed to help.

Mrs. E. F., was a 72-year-old poorly nourished white woman, who was seen in August, 1951, complaining of severe pain and diminished vision in the left eye for two weeks. Photophobia, lacrimation, and moderate conjunctival injection were present but no discharge was noted. Gross inspection showed an elongated gray area of infiltration extending from the 7-o'clock to 9:30-o'clock positions and about three mm. toward the corneal center. There was a trough or gutter along the corneal margin corresponding in position to an arcus senilis.

Under slitlamp microscopy the remainder of the cornea was clear with no keratic precipitates. No cells or flare were present in the anterior chamber and the iris; lens and vitreous were normal. With the slitlamp beam it was apparent that a trough or gutter in the corneal margin was situated approximately one mm. from the limbus. The digital tension was normal. The cornea was not suitable for tonometry. The fundi were normal.

The usual treatment of topical atropine

(one percent, three times daily), sodium sulfacetamide (15 percent every hour), heat, foreign protein, and natural vitamin-A therapy failed to halt the progress of the ulcer. The patient complained of incessant pain unrelieved by the ordinary analgesics. A smear and culture were negative for bacterial growth.

In two weeks the ulcer had spread to the 11-o'clock position on the periphery and was encroaching on the pupillary area of the cornea. The excavated peripheral zone appeared to be ready to perforate and the patient pleaded for enucleation to rid her of the pain.

On August 16, 1951, the cornea was covered with a total conjuctival flap. The excruciating pain continued unabated and it was apparent that the ulcer was increasing in size. On September 4, 1951, the conjunctival flap was removed and a penetrating keratotomy was performed from the 11-o'clock to 7-o'clock position. The incision was reopened on two successive days and aqueous was permitted to escape. Nothing was done to the eye on the third day and by the fourth day the incision could not be reopened easily at the bedside. The pain continued and the ulcer spread along the inferior margin toward the 5-o'clock position.

On September 14, 1951, a second keratotomy was performed from the 7-o'clock to 4-o'clock position. The pain lessened and now stayed controlled with Demerol which previously had little effect. Under tetracaine anesthesia, the wound margin was depressed and the anterior chamber emptied daily for five days. The eye became comfortable at once and the patient was discharged on topical atropine and haliver oil capsules. All medication was discontinued in one month.

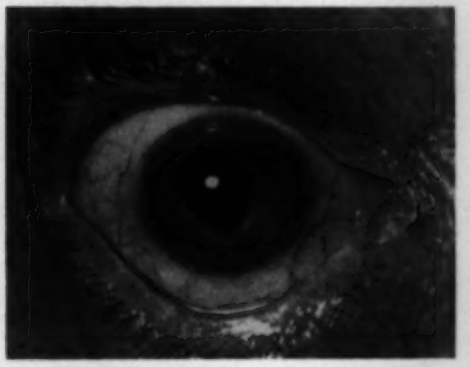

Fig. 1 (Harley). Right eye. Appearance of the cornea four months after penetrating keratotomy. Present corrected vision, right eye, 20/30.

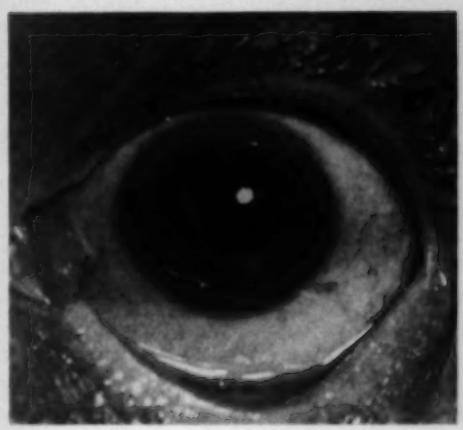

Fig. 2 (Harley). Left eye. Appearance of the cornea 21 months after penetrating keratotomy. Present corrected vision, left eye, 20/70.

The vision gradually cleared and by February, 1952, the acuity in the left eye had improved from counting fingers at two meters to 20/70 with -1.0 D.sph. $\bigcirc$ $+1.75$ D.cyl ax. 145°. The patient was urged to improve her nutrition and take supplementary vitamins.

The patient remained perfectly well and comfortable for 16 months and then in January, 1953, she developed pain in the right eye. Believing that it was similar to the trouble she had had with the left eye, she began using the remaining portions of her old medicines. After 10 days she could not stand the pain any longer, and applied for help.

A typical elongated, grayish area of infiltration was present from the 7-o'clock

Fig. 3 (Harley). Diagram of penetrating keratotomies. Right eye on the right side.

to 11-o'clock positions. A furrowed area was beginning to become prominent and there were areas of infiltration in front of the undermined, overhanging border. The lesion resembled in every detail the ulcer that had been studied and treated in the opposite eye.

The first culture and smear taken was reported "no growth." The second culture was reported as Staphylococcus albus but was coagulase negative and probably not pathogenic.

With the previous experience fresh in mind, a delimiting keratotomy from the 12-o'clock to 10-o'clock position and then from the 10-o'clock to 6-o'clock position was performed on February 9, 1953. The wound edges were depressed daily for five days and aqueous was allowed to escape. Supportive therapy with intramuscular milk injections and topical sulfacetamide (15 percent) was used. She was discharged on February 16, 1953, and appeared much improved. However, during a week's absence, the grayish zone of infiltration began spreading along the limbal border in the opposite direction toward the 3-o'clock position. She was readmitted to the hospital February 21, 1953, and a keratotomy was performed from the 12-o'clock to 3-o'clock position and from the 3-o'clock to 6-o'clock position, approximately four mm. from the limbus. One mm. at each end separated the former incision from the new keratotomy. Aqueous fluid was released daily, under local anesthesia, by depressing the apex of the corneal incision. Supportive therapy in the form of intramuscular milk and Demerol was used. The patient was discharged February 28, 1953, with a comfortable, healing eye.

In July, 1955, the vision in the right eye was 20/30 with +1.25 D.sph. ⊂ +1.0 D.cyl. ax. 165°. She is able to read and sew with complete comfort.

Comment

The patient has been followed for three years and the best corrected vision of the right eye remains at 20/30 and the left eye at 20/70. There have been no signs of recurrence and the patient has been comfortable. The better visual result in the right eye is attributed to the fact that no time was lost in the use of anibiotics and a conjunctival flap, as occurred in the first eye. The refractive error prior to the development of the corneal ulcers was available. It is interesting to note that the amount of astigmatism remained unchanged following the keratotomy, although the axis shifted 90 degrees in the left eye.

Since the diagnosis of Mooren's ulcer is made on its clinical appearance, it must be differentiated from other marginal ulcers of the cornea. Infiltration and ulceration of the corneal margin in catarrhal conjunctivitis, ring ulcer of the cornea, phlyctenular keratitis, rosacea keratitis, and trachoma usually offer no difficulty in recognition when the fully developed disease process is present. The marginal ulcer of Zur Nedden and the superficial marginal keratitis of Fuchs may resemble Mooren's ulcer in the initial stages. However, these ulcers are more superficial and benign and tend to get well, as a rule, without visual loss. There are no deep furrows or overhanging necrotic edges along the ulcer margin, which is characteristic for chronic serpiginous ulcer. In Mooren's ulcer the pain is more severe, the process progresses more slowly,

and the prognosis for vision is often poor. The characteristic healing by epithelization from the limbus toward the center of the cornea does not take place until vascularization has occurred.

Summary

1. A case of bilateral Mooren's ulcer (chronic serpiginous) was successfully treated by delimiting keratotomy. Involvement of the second eye took place 18 months following the attack in the first eye.

2. The production of hypotony over a period of time seems to offer good results. This can be accomplished by a delimiting keratotomy, repeated paracenteses, or making a fistulous track with a cautery.

3. A ring-shaped, lamellar, corneal graft has been reported as giving good results and may be used prior to a keratotomy.

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SLITLAMP MICROSCOPY OF THE VITREOUS AND THE FUNDUS*

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The slitlamp examination of the vitreous and of the fundus enables us to understand better certain pathologic pictures and frequently facilitates the differential diagnosis between ophthalmoscopically similar pictures. Since more details are seen, pathologic changes are discovered earlier. It would also seem to be worthy of mention that this method allows the stereoscopic examination of the fundus with the help of a cheap supplementary device to the slitlamp, an examination for which Gullstrand's expensive binocular ophthalmoscope was formerly used. But the slitlamp examination of the fundus is not only a substitute for binocular ophthalmoscopy. It also gives more information than the latter.

Three things distinguish the examination of the fundus with the slitlamp from the usual ophthalmoscopy:

1. The fundus is illuminated focally by means of an image of a variable slitdiaphragm.

2. The illuminating beam and the direction of the examination form an angle.

3. The examination is made with the aid of a binocular microscope and is stereoscopic.

Only the application of all three principles together may be termed slitlamp microscopy of the fundus.

The history of this method of examination goes back to Köppe,[1] who determined the principles of the technique which is still in use today, with a special contact lens for the elimination of the corneal refraction, a silver mirror for reducing the angle between illumination support and microscope, and a binocular microscope, Valois and Lemoine[2]

* Presented at the International Symposium on Physiology and Pathology of the Eye, at Iowa City, September, 1954.

then demonstrated that, instead of a contact lens, a concave lens of 55D. may also be used, being placed in front of the eye. Friedenwald[3] suggested a direct ophthalmoscope containing a device for oblique focal illumination. Zamehof[4] invented a supplementary device to Gullstrand's ophthalmoscope, which projects onto the fundus a slit image which can be seen binocularly and stereoscopically without the aid of a contact glass or a lens in front of the eye. Busacca[5] and I[6] have improved on Köppe's technique, Hruby[7] on that of Lemoine and Valois.

Let us now give a brief description of our method, by means of which details in the vitreous and fundus may be seen especially clearly. Maximum mydriasis is important if the periphery of the fundus is also to be examined. This is achieved by instillation of homatropine and Glaucosan or by paralimbar injection of a drop of procain-adrenalin solution. For anesthesia 0.2-percent Novesin is used.

Then a contact lens with a flat anterior surface is placed on the eye for the examination of the axial parts of the vitreous and of the posterior pole of the eye. Methocel is used as the liquid filling the space between contact lens and cornea. The contact lens has a funnel in front, which serves on the one hand to hold the contact lens in order to eliminate by means of slight turning and tilting reflexes or astigmatism, and on the other hand to keep the surface dry. A reduction prism enables the angle between the illuminating device and the microscope to be reduced.

For the examination of the vitreous the largest possible angle between the illuminating arm and the microscope should be chosen. For the examination of the fundus the angle chosen must be acute. If the angle is zero degrees, the result is binocular ophthalmoscopy in focal illumination, but no longer slitlamp microscopy. Only if the largest possible angle is used for the examination of the vitreous does the light reflected by the fundus cause the least possible disturbance during focal examination.

The examination of the vitreous and fundus with slitlamp is facilitated considerably by a fixation point for the other eye. For the examination of the periphery of the fundus and vitreous I have a contact lens that, like my gonioscopic lens, contains mirrors, to be exact three mirrors for various zones of the periphery of the fundus, from a distance of approximately 30 degrees from the fovea to the outer periphery. With this contact lens a binocular examination of only the upper and lower periphery is possible. In order to render visible the lateral parts of the periphery of the retina, the patient must lie on his side on a couch and the eye is examined in this position.

The reason why the lateral parts of the periphery cannot well be examined directly is that the pupil, deformed by perspective to a vertical oblong or lengthened oval, does not leave room for the beam of light and the two pencils of rays to the binocular microscope to pass through together side by side. With the examination of the upper or lower periphery, on the contrary, such an examination is easily possible through the pupil which appears in perspective as a horizontal lengthened oval. An attempt was made to save space by examination of the lateral parts with a horizontal illumination slit. However, with a horizontal slit the conditions for a stereoscopic examination of the optical section are, for physiologic reasons, far less satisfactory than with a vertical slit.

Thus the examination of the periphery of the retina is more complicated than that of the central part of the retina, and, therefore, this examination should be made only after previous ophthalmoscopy; it must first be determined which point of the periphery it is desirable to examine thoroughly, and this spot must then be sought out with the slitlamp.

For all examinations of the vitreous it is important that the examination should be made in the optical section and by reflected

light. The retina is to be examined in the optical section and next to it, where the light diffused from the choroid illuminates the retina from behind. The first method renders it feasible to determine accurately the spatial relations of changes, the second enables the finest optical differences to be distinctly seen. With an enlarged pupil it is easily possible, by means of slitlamp whose light pencil and microscope are adjusted to a point on the common axis of revolution and by coupled turning of the two together, to bring into view stereoscopically the place to be examined. The stereoscopic viewing is essential in the examination of vitreous and fundus.

When using the Hruby lens, special care must be taken that the glass is placed as near as possible in front of the eye, centered to the microscope and to the center of rotation of the eye. Field of vision and magnification are smaller with the Hruby lens than when a contact lens is used. On the other hand, with recently operated and sensitive patients the use of the Hruby lens placed in front of the eye is to be advised.

Certain deceptive pictures from the reflecting surfaces of the eye that seem to lie near the center of the vitreous will soon be recognized, and may be avoided by tilting the contact lens; in the same way, astigmatic distortion of the illuminating beam may be reduced to a minimum by shortening the slit.

For the examination of the vitreous one proceeds, as has already been stated, by making the angle between the illumination support and the microscope at first as obtuse as possible, after the contact lens or Hruby lens has been placed in position in front of the eye. The light should be as strong as possible, for the examination of the vitreous is in fact a kind of ultramicroscopy.

Parallel to the lens equator on the far periphery of the posterior surface of the lens a reflex line may be seen, which indicates a slight angle in the curve of the posterior surface of the lens. It is the point where the anterior membrane of the vitreous curves backward from the lens, the hyaloideocapsular ligament of Wieger. Almost immediately beyond it the zonula fibers begin. Sometimes the hyaloideocapsular ligament may remain intact even after an intracapsular cataract extraction and then zonula fibers may be clearly seen entering the vitreous on its periphery.

The rest of the hyaloid membrane normally lies so close to the lens that it cannot be seen. Only after trauma or in individual cases of old people is it detached from the lens and is seen as a smooth membrane, as Weber[8] has pointed out. Otherwise an optically empty space is seen behind the lens, behind which lies the membrane plicata, the boundary of Cloquet's channel, better called the tractus hyaloideus (Szent György). It is attached to the anterior membrane of the vitreous in a circular line of some five mm. in diameter, that is, apparently on the posterior surface of the lens, and must not be confused with Vogt's reflex-line, which is much smaller, and which surrounds the anterior rudiment of the hyaloid artery.

In the optical section the upper boundary of the canal of Cloquet is seen like a membrane hanging down vertically in folds. Approximately four mm. from the axis below, it curves around toward the back. The lower boundary leaves the posterior surface of the lens about two or three mm. away from the axis and also runs first downward and then toward the back. The narrowed channel then continues nasally downward in the form of a transverse slit and rises again in about the middle part of the vitreous to a point above the axis.

Here its boundary is again seen as a plicated membrane; then it passes in front of the macular region mostly upward temporally and then curves backward and downward and nasally in the direction of the disc. It often contains at the most varied places filifoam remains of hyaloid artery. The plicated membrane is accompanied by alternately optically denser and more transparent layers

of the vitreous, which appear as bands in the section. The whole begins to sway and tremble violently at the slightest movement of the eye, so that it is very difficult to follow up the structure.

The further one penetrates into the vitreous, the more acute must the angle be between the illumination support and the microscope. Presently the fundus is seen to interfere by its red light and this renders the examination of fine details of the vitreous impossible. Nevertheless, the beam of light can be so directed that most of the light from the fundus is concealed by the iris. The use of a short slit often facilitates the examination.

The most important pathologic changes in the vitreous are, aside from the embedding of blood and exudate cells, vitreous detachment, fibrous vitreous destruction, and membrane formation in the vitreous space. However, before discussing these changes, I should like to give a brief description of a rare finding that may cause errors in diagnosis: a vitreous cyst. It may easily be mistaken for a parasite, for instance an echinococcal or cysticercal cyst. In the microscopic examination, however, inflammatory symptoms and the characteristic scolices are lacking.

The detachment of the vitreous, which with the ophthalmoscope can only be recognized by the characteristic prepapillary opacity, may be seen clearly and distinctly with the slitlamp. It is very frequently found in older people.

The vitreous layers situated behind the lens are seen to be more dense and behind them comes a wavy membrane, which is in constant though slight movement. Behind this again, toward the retina, there is an optically empty space. This posterior boundary of the detached vitreous first passes from above parallel to the posterior surface of the lens. It then curves toward the back and approaches the retina which it usually joins a little below the disc.

In some cases it can be seen that it is detached from the retina also in the lower parts. Often it cannot be determined whether the vitreous is still firmly attached to the retina in the lower parts, or whether it is only the force of gravity that has caused the thickened vitreous to fill completely the vitreous space below but not above. Only where a hemorrhage has occurred between vitreous and retina does a streak of blood in front of the retina indicate where the detachment ends.

In front of the region of the disc in the posterior surface of the vitreous detachment an opaque thickening is always found, often like a ring, sometimes only like a twisted strip or row of beads and fibers. It is usually assumed that the phenomenon in question is a thickened zone of the vitreous normally situated on the edge of the area of Martegiani. Much may be said in favor of this opinion, but whether this is really always the case, or whether it is after all related to the vascular funnel, as Busacca assumes, I will not venture to determine. At any rate, I shall show you here a picture in which this opacity is connected with the vascular funnel; afterward you will see a case where it corresponds quite definitely to the edge of the area of Martegiani.

The upper end of the detachment is the most interesting because in the opinion of many authors the formation of holes in retinal detachment is connected with its configuration. In a large number of cases (50 percent) the detached vitreous may be seen in the upper periphery to curve backward and to be lost from sight after running more or less parallel to the retina. In other cases, the upper end of the vitreous detachment lies so near to the ciliary body, at or in front of the ora serrata, that it cannot be seen at all (20 percent). Finally, there are cases where the thickened vitreous part comes to an end behind the ora serrata, adhering more or less vertically to the retina (30 percent). In these cases it is understandable that the constant pull of the vitreous might in time lead to retinal degeneration. However, hitherto I

have been unable to follow up the development of such changes in the case of a vertical adhesion of the vitreous detachment to the retina.

If, on the contrary, the behavior of the vitreous in cases of retinal detachment is examined: firstly, a detachment of the vitreous will always be found; and secondly, in addition in most of the cases, a fibrous destruction of the detached virteous was also seen.

A mass of fiber spread through the detached vitreous, with different degrees of thickening at various points. The vitreous adheres by such fibers to the holes and tears and to Blessig's spaces. I shall speak of this later.

In one case of extreme malignant retinal detachment in a 15-year-old patient, in whom there occurred first a total disinsertion at the ora in the right eye, followed one year later by a disinsertion in the left eye, I observed the following:

At first there was a network composed of thick, rather strongly refractile, vitreous fibers behind the lens, with tension toward the retinal periphery. Then half the circumference of the retina tore off nasally and although an operation was performed an hour after the examination, the detachment had meanwhile advanced as far as the disc. A barrage was able to prevent the detachment from progressing further for a period of four weeks. Then, in the course of one night, the detachment became total. Finally, thick glassy vitreous threads were seen to attach themselves everywhere to the detached retina like suckers.

It is obvious that the vitreous could be only imperfectly examined in the early stages in this case, so that I am unable to do more than hazard a guess concerning the mechanism of the detachment in this case. The extremely fibrous change of the vitreous framework with subsequent shrinkage, but without detachment from the retina, would seem to me to be the cause of the deleterious progress of this case.

Detachment of the vitreous does not only occur spontaneously and especially frequently in cases of advanced myopia and in old age, but also accompanies very many intraocular diseases. Uveitides, periphlebitis, degeneration of the posterior pole of the eye due to old age, injuries by perforation, and vitreous hemorrhages, are accompanied by detachment of the vitreous. In cases of inflammatory changes it is not unusual to find precipitatelike deposits on the posterior membrane of the detached vitreous. In inflammatory processes the vitreous humor is richer in albumen which may be recognized by a distinct Tyndall flare in the vitreous, both where its framework exists and in the otherwise optically empty region.

From the fibrous destruction we come to membrane formation in the vitreous, which is observed especially frequently following hemorrhages, but also without them. We saw, for instance, in an initial stage of von Hippel-Lindau's disease such membrane formations without ingrowth of vessels in front of the posterior pole. These membranes are especially pronounced when vessels form in them, as in proliferating retinopathy. It must be particularly stressed that in the vitreous space there are newly formed vessels only within membranes. Free new growth of vessels in the mass of vitreous itself is never seen. We (Bangerter-Blaser[9] and Hruby[10] were of the opinion that these membranes are the surface of the detached vitreous. It is certain that they form a surface of the vitreous, since they spring from the retina with its vessels. I am not sure, however, whether it is always the preformed surface of the detached vitreous that the membranes follow, as many-layered membranes can now and again be seen.

Thickened inflammatory membranes sometimes exhibit striking window formations, through which the fundus may be seen quite clearly.

Now let us turn to the changes in the periphery of the retina. The ora serrata can generally be made visible only in cases with

an iridectomy or by pressure on the region of the ciliary body and indentation of this part. Here we see a picture of Iwanow's spaces as a series of glassy blebs on the ora serrata. Otherwise the normal periphery exhibits no peculiarities. In this region, however, layer holes in the retina do occur as pathologic changes which are clinically unimportant. Weber[8] was the first to describe them. Here you see pictures of these interesting changes, the holes have the appearance of having been punched out or perforated, just as do the so-called macular holes. Their genesis we do not know; in the cases examined by us they did not lead to detachment of the retina. Possibly they developed from cysts.

The most important findings in the periphery of the retina arise in cases of retinal detachment. In the first place, slitlamp microscopy allows us to distinguish quite clearly between attenuated parts of the retina and holes. You know how difficult this often is by ophthalmoscopy. Over attenuated places the slitimage can be seen without interruption; where there are holes it is interrupted. It is not unusual to see a minute hole in a larger extremely thin retinal area. There seems to be no detached retina without detached vitreous. Even more rarely does an idiopathic retinal detachment seem to occur in cases of detached retina without—at least local—fibrous degeneration.

So far I have always found vitreous adhering to the holes and degenerated spaces (Blessig's spaces) in the form of threads and delicate strands. In most instances they seemingly disappear some distance from the tear. In the cases of holes with an operculum fibrous masses of vitreous adhere to the operculum. In the region of the holes, beside Blessig's spaces, to which vitreous also adheres, a pronounced cystic edema of the retina is often found, which is only seen well in reflected light. Cystic edema is otherwise found only at the posterior pole of the eye. Round layer holes that look as if they had been punched out are, as we know, also a characteristic feature of the fovea, but both types of change are also met with in the periphery of the retina. In this respect, periphery and center are analogous.

If, in the cases of Blessig's degeneration with adhesion of vitreous strands, the formation of tears with vitreous fiber at the edge or on the operculum indicates the genesis of the hole formation, the slitlamp examination of many cases of detachment can take us further and supply important indications for the treatment. This is shown by the following case history:

I saw the patient for the first time in February, 1945. He had suffered from repeated vitreous hemorrhages in the right eye; now a detachment of the retina had appeared. On examination a vitreous detachment was found above, and a fresh flat retinal detachment below. Above, in a few places, blood and remains of vitreous could be seen adhering to the retinal vessels. In the area of the detached retina an extensive horizontal blood line could be seen. Immediately above this line of blood lay a triangular retinal tear.

With the slitlamp it was seen that the blood line marked the point down to which the vitreous had become detached from the retina. Below the blood line the two were connected, above they were separated. The blood had gathered in the pocket between the two and thus formed the transverse streak of blood.

The vitreous detachment extended only as far as just below the retinal tear. From there the vitreous and retina began to be detached together. The mechanism of the repeated hemorrhages and the formation of holes was as follows:

As is usual, the vitreous detachment had begun at the top. This process had led to lesions of the vessels accompanied by hemorrhages, as is often seen in elderly people; indeed we know that a detached retina frequently begins with hemorrhages of the vitreous. The vitreous detachment advanced,

causing slight hemorrhages from time to time, and finally causing a tear in the retina, but this was only after the vitreous detachment had already advanced as far as the lower parts. From this moment onward the vitreous did not become further detached from the retina but retina and vitreous together became detached from the choroid. The rest of the history of this case justifies this conclusion.

The hole was coagulated diathermically and the patient kept in bed in a semisitting position in accordance with Gonin's procedure. The retina did not become attached. We came to the conclusion that the cause of the failure was that, on account of the upright position of the patient, the vitreous had dropped down dragging the retina away from the choroid. Therefore, after a second surgical intervention at the same spot, the patient was made to lie flat on his back, so that the vitreous now pressed the retina against the posterior wall of the globe whereupon the detachment healed.

Very instructive also are two further cases in which we could find no hole, in spite of the most careful ophthalmoscopic examination. With the slitlamp examination a tiny hole was discovered, about one disc diameter away from the papilla just at the edge of the area of Martegiani, where the canal of Cloquet is usually attached to the retina. This should be noticed. Without the slitlamp these holes cannot be seen at all. Minute retinal holes seem to have a predilection for the edge of the area of Martegiani.

It is interesting to note that in certain cases of cyclodialysis with very soft eyeballs the peripheral flat choroidal detachment may be seen with the slitlamp, in good agreement with the histologic picture.

Of the findings at the posterior pole of the eye, only those deserve our attention in which slitlamp microscopy is more efficient than the usual ophthalmoscopy, or which bring to light new facts. For, whereas gonioscopy is not possible except with the aid of a contact glass and a microscope, we have in ophthalmoscopy an old, excellent, and simple method of examining the fundus.

The slitlamp examination of the disc shows us its normal relief and any differences in level are seen just as well as with Gullstrand's binocular ophthalmoscope. On the other hand, we cannot endorse the statements of Köppe and others that a certain differential diagnosis between optic neuritis and the early stages of papilledema or choked disc is possible with the slitlamp. In both diseases, detachment of the membrana limitans can be seen. Nor is the levelling of the central depression of the disc a distinctive sign. Even magnified 20 times, the perivascular lymph spaces cannot be seen, which, according to Köppe, are said to be distended in choked disc. Nevertheless, in cases of genuine optic neuritis an increased vitreous flare is often observed in front of the disc or fine precipitates are noted on the posterior surface of the detached vitreous.

In one disease of the optic nervehead, the slitlamp examination is of great value. It permits the differential diagnosis between drusen situated deep down in the disc and optic neuritis. Both changes are accompanied by blurred edges of the disc and visual field defects. If, however, a sharply focussed pencil of rays is thrown on the edge of the disc, the drusen are seen in the tissue of nervehead as more strongly lighted refracting transparent bodies, shining in the reflected light. In cases of neuritis nothing of this kind is seen or only a bright line around the papilla apparently situated in deep layers of the retina, and this is often particularly distinct in cases of papilledema. I do not know the anatomic basis of this phenomenon.

I do not need to speak about the recognition of certain malformations, pits in the nervehead and colobomas of the retina and the choroid, but there exist rudimentary colobomas represented by a slight depression of the fundus below the somewhat

asymmetrical but otherwise normal disc, without visible change of structure of the choroid (v. Szily's astigmatismus fundi[12]). A patient with such an anomaly was sent to us from another clinic as being a case of optic neuritis because a scotoma had been found above the blindspot on the Bjerrum screen. As the indented part exhibited only scanty retinal vessels the difference of level had been overlooked with the ophthalmoscope. The scotoma turned out to be the consequence of the difference in refraction; when the circumscribed myopia of 6.0D. was fully corrected, the scotoma disappeared.

Although the diagnosis of medullated nerve fibers causes no difficulties if the picture is typical, single medullated fibers can often only be distinguished with the slitlamp from myelin fiber degeneration by their sharply cut contour.

Normally during the slitlamp examination, the thickness of the retina is seen in the section, and it is greatest temporally from the disc and in the perifoveal wall. The normal fiber pattern is very clearly seen with focal illumination. In cases of atrophy of the papillomacular bundle the retina is attenuated between nervehead and macula and the nerve fibers are lacking there. But even circumscribed atrophy of the retina may be recognized in the section in the region of the retinal pole. I saw, for instance, a patient who, a month previously, had noticed during a period of military service that the sight of his left eye had suddenly deteriorated. My examination showed a paracentral absolute scotoma accompanied by normal sight. Nothing could be seen with the ophthalmoscope. The slitlamp examination showed a circumscribed attenuation of the retina in the region of an apparently normal cilioretinal artery. It must therefore have been a case of a temporary block of this cilioretinal vessel.

The normal depression of the fovea is clearly seen with the slitlamp as is the yellow spot whose brightness and size Busacca[13] saw changing during the actual examination. The pathology of this region appears particularly distinct during a slitlamp examination.

A disease which frequently gives rise to diagnostic errors is the chorioretinitis centralis serosa. It is either mistaken for a retrobulbar neuritis or not recognized at all. When the disease is situated just in the macula, a distinct bulging of the peripheral parts may be seen, with a navel-like depression, corresponding to the fovea, so that the optical section of the surface of the retina is shaped like a Cupid's bow.

In the majority of cases the retina is not more opaque than normally but its posterior limit cannot be clearly seen; yet fluid must lie between retina and choroid membrane. For small white spots are seen at a distinct distance in front of the choroid behind the retina, evidently situated on the invisible posterior surface of the retina. In front of the edematous retina there is a delicate vitreous detachment, which it is often not easy to prove. At first the aspect of the choroid seems to be unchanged, later it becomes slightly marbled.

Because of the presence of a relative central scotoma accompanied in general by slightly decreased visual acuity, this disease is often mistaken for retrobulbar neuritis. With the ophthalmoscope a more strongly refracting line of demarcation is seen around the edematous zone, a phenomenon that one must keep in mind in order to be able to make a diagnosis from the ophthalmoscopic picture. Subjectively, the scotoma is often yellowish and especially clearly observed on the patient's awakening. There is a slight metamorphopsia, which may easily be demonstrated with Amsler's net. During the disease the refraction is more hypermetropic than it is before or after.

All these characteristic features are much less pronounced when the changes are situated outside the fovea. Then the patient's complaints are not characteristic: dimmed sight, perhaps slight distortion of straight

lines. In such cases the diagnosis can seldom be made without the slitlamp.

Usually nervous disorders are assumed, especially because the change is found in vasolabile individuals. The etiology of the disease is unknown, but vasolability seems to have a part in it. In our country the disease is not so rare, but it is often not diagnosed.

Although in chorioretinitis centralis serosa there is a circumscribed retinal detachment accompanied by a discrete retinal edema, the central retinal edema is in most cases characterized not only by thickening of the retina but also by cyst formation. This edema is very frequent, without other anatomic concomitant symptoms as the first symptom of vascular disorders, especially in elderly people; as concomitant symptom, for example, in thrombosis of the central vein, in retinal detachment, in senile disciform macular degeneration, in retinitis circinata, or uveitis.

This edema, which often cannot be seen with the ophthalmoscope and even in red-free light can only be recognized with difficulty, reduces the visual acuity. It is capable of being cured.

The most serious forms of this edema are found in circinate retinopathy in which the edema comprises the whole area within the wreath of yellow spots. The cysts lie at any depth in the retina, and in focal light have a honeycomb appearance. In indirect lighting they are seen as small shining bubbles, when they are biggish; if they are tiny, the retina assumes a hammered appearance. The largest cysts are always to be found in the fovea. A single cyst can give the impression of a macular hole in an ophthalmoscopic examination. With the slitlamp, however, it is easy to distinguish a hole in the macula from a macular cyst. The cyst has an anterior wall, which can be seen quite well in the optical section, the hole has none, and the latter has also mostly thickened edges and is always accompanied by a detached vitreous, in which the operculum of the hole may occasionally be found.

Besides the cystic edema there are two other edema formations that would seem to be important. In both a moderate increase in the thickness of the retina is found. Both are connected with vascular changes, mostly in the form of capillarosis spots, of which I have yet to speak. Sometimes capillary aneurysms are also found. One form of edema is found especially in cases of malignant hypertension and thrombosis of the central vein. It is cloudy, so that the choroid can be recognized only with difficulty. Besides capillarosis spots, streaky hemorrhages and cotton-wool patches are mostly present. In cases of diabetic retinopathy, with arteriosclerotic changes at the posterior pole, another quite glassy transparent edema is seen, characterized by the fact that the shadow of the retinal vessels can be clearly seen on the choroid.

The differential diagnosis between capillarosis spots and small calcified "drusen" is often difficult. Capillarosis spots were considered by French authors to be a sort of "etat crible" of the retina. That they assuredly are not, but they are various types of degenerative foci of vascular origin in the retina, sometimes the residues of slight hemorrhages, sometimes accumulations of fatty granule cells, sometimes hyaline degeneration of capillary aneurysms. They always indicate damage of fine retinal vessels.

Drusen, on the other hand, are a thickening of Bruch's membrane, probably often the results of arteriosclerotic changes in the choroid. Only when they appear in large numbers close to each other are they harmful to the sight.

Capillarosis spots are found in the thickness of the retina and are always cloudy white to yellowish-white. In the lightbeam of the slitlamp their position in front of the choroid is easily seen; sometimes they even cast a shadow on the choroid.

Drusen lie in the choroidal surface, are mostly yellowish and transparent and ex-

hibit, when they are fairly large, a golden rim reflex in indirect illumination. Calcified drusen are whitish and only their position on the surface of the choroid make them distinguishable from capillarosis spots.

It is of importance for a prognosis to distinguish between retroretinal and intraretinal hemorrhages, because the former often, though not always, heal up with an almost complete restitution of the sight, while the latter do not.

And now a few words on diseases of the choroid. A peculiar change in the surface of the choroid is sometimes seen in the early stages of senile macular degeneration, frequently together with cystic edema: the surface of the choroid membrane, which is normally smooth, is roughed, as if it had been treated with emery-paper.

In cases of choroiditis, some important findings can be made with the slitlamp.

1. Every fresh focus of choroiditis is accompanied by an edema of the retina, which can be easily recognized with the slitlamp as a cloudy thickening of the retina even in the bewildering mass of a disseminated choroiditis of long standing.

2. In solitary choroiditic scars of the type of juxtapapillary choroiditis, smallish cloudy looking foci, sometimes reminiscent of precipitates, not rarely remain behind and may be followed by relapses. So long as their presence can still be detected with the slitlamp, a relapse is possible. Burned out choroiditis foci are entirely sunk in.

3. In a case of sympathetic ophthalmia with central scotoma we saw only a slight retinal edema with the ophthalmoscope. With the slitlamp not only was there a cloudy edema of the retina but behind it a yellowish cloudy thickening of the choroid as a sign of the fresh diffuse sympathetic choroiditis, which, as we know, cannot be seen with the ophthalmoscope.

Choroidal sarcomas are especially easily recognizable with the slitlamp, in most cases even if the retina over them is totally detached. When magnified slightly the tumor is seen through the detached membrane. A flat tumor beside the disc, with very severe cystic retinal edema over it, rouses strong suspicions of an angioma of the choroid.

Finally, it must be mentioned that it is often possible with the slitlamp to distinguish the tail of a foreign body situated in the wall of the globe from a clot of pigment.

I have now reached the end of my report. Whatever holds good for gonioscopy also applies to the slitlamp examination of the fundus and vitreous. It is a method which can be turned to good account only in combination with the other forms of examination. It provides valuable supplementary information where ophthalmoscopy fails, but anyone who has made many slitlamp examinations of the fundus will find that his eye is sharpened for opthalmoscopy too, and even with the ophthalmoscope he often recognizes changes, the interpretation of which he owes to the slitlamp examination. The fundus should never be examined with the slitlamp without accurate preliminary ophthalmoscopy. Interesting places which it is difficult to interpret may then be sought with the slitlamp. It goes without saying that a systematic examination should never be omitted. In this way, this method may be used to its greatest advantage. Much, however, still remains to be explained. Above all, the relation between the biomicroscopic findings and the histologic picture still remains to be explained in many cases.

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POSSIBLE ANTIVASCULARIZATION FACTORS IN THE CORNEA*

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All blood vessels arise embryologically as a differentiation of mesenchymal tissue. Three stages in the development of the vascular system were postulated by Roux[1]: first, the stage in which the formation of vessels depends entirely on genetic factors; secondly, a transitional stage in which hereditary formation is gradually supplanted by adaptational factors; and the final stage, where the differentiation of vessels is controlled by the hemodynamic factors of the circulation. Once circulation has been initiated, any further extension is brought about by a sprouting of pre-existing capillaries, since vascular endothelium can no longer be formed by the differentiation of mesenchyme cells but only by the proliferation of already differentiated endothelial cells.

The extension of new capillaries by the formation of vascular buds is an essential process in the adult for the adjustment of the peripheral circulation to varying functional demands and in the vascular reactions to inflammatory stimuli. It has been suggested that the formation of new capillaries in the adult may be related to the metabolic activity of the endothelial cells of the capillary vessels already in existence, as shown in the vascular response to an inflammatory focus where the increased metabolic activity of the tissues involved must also involve a similar response in the capillaries in the immediate neighborhood.[2]

The normal cornea is a completely avascular tissue. Vascularization of the cornea represents a pathologic process which can be induced by local disease or injury, as well as by more generalized disturbances, such as nutritional deficiencies. It seems probable that the basic common denominator of the factors which give rise to corneal vascularization is a disruption of the delicate balance of the metabolism which maintains the functional and structural integrity of the cell. The increased nutritional needs for the process of repair may require an increased blood supply leading to a vascular invasion of the cornea.

Possible factors responsible for the primary stimulus to vasculogenesis of the cornea have been attributed to edema of the corneal stroma lessening the physical resistance to growth of new blood vessels,[3] the presence of a diffusible vasogenic factor in the tissue,[4] and to the enzymatic hydrolysis of the mucopolysaccharides of the ground substance of the cornea.[5] Facts which favor the latter hypothesis are that structures with a high mucopolysaccharide content, including the cornea, cartilage, and the ocular vitreous, are devoid of blood vessels. Furthermore, when the mucopolysaccharide content of the cornea is reduced by the injection of hyaluronidase into the aqueous or corneal tissue, vascularization is produced.[6]

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There is also the known decrease in mucopolysaccharide content of the cornea after alkali burns.

On the basis of these observations, it was decided to investigate the inhibitory effects, if any, of the administration of purified preparations of the corneal mucopolysaccharides on vascularization of the cornea.

Mucopolysaccharide fractions were isolated according to the procedure of Meyer et al.,[7] using lyophilized bovine cornea* as the starting material.

The four fractions tested for activity were Fraction 1, corresponding to chondroitin sulfate, Fractions 2 and 3, resembling chondroitin, and Fraction 4B, a sulfated mucopolysaccharide, composed of equimolar quantities of glucosamine, acetyl, galactose, and sulfate, for which Meyer proposed the name keratosulfate. This last compound represents approximately half of the total mucopolysaccharide content of the cornea, and, to date, has been found only in the cornea and in growing bone.[8]

Prior to use, the calcium salts of the various fractions were converted to the sodium salts by passage through the sodium form of the cation exchange resin, Amberlite IR-120. The filtrates were lyophilized, made up to the desired volumes with physiologic saline, and the solutions flash pasturized at 80° C. for three minutes before injection.

Method

Five groups containing six albino rabbits each were used. Each group sustained corneal burns equally on both eyes with either sodium hydroxide or sulfuric acid under topical pontocaine anesthesia. The alkali burns were produced by injecting 0.02 ml. of 0.5N sodium hydroxide into the superficial corneal layers about 2.5 mm. from the superior limbus, using a 26-gauge needle; the acid burns were inflicted by the application of a swab wet with 30-percent sulfuric acid onto the surface of the cornea three mm. from the superior limbus for 30 seconds. The magnitude of the resultant vascularization, which began to appear in about three to four days, was more consistently reproducible when the acid burning technique was used.

In one eye of each rabbit in four of the groups, 0.05 ml. of a 10-percent solution in saline of the appropriate mucopolysaccharide was injected subconjunctivally at the point nearest the burn. The fifth group of animals was treated with a heavier dosage of keratosulfate (Fraction 4B), namely 0.10 ml. of a 20-percent solution being injected. Aureomycin ointment was used in the conjunctival sacs on the operative day. Re-injections of the mucopolysaccharide fractions were done on the third and fifth days. All eyes were examined daily with a loupe during the first week and at longer intervals thereafter.

Results

The results were measured by the number and appearance of the blood vessels invading the cornea, slitlamp examinations being performed when necessary. No significant differences were found between the treated and the control eyes over a period of four weeks.

Discussion

The experiments reported here indicate that there is no evidence for antivascular properties of the component mucopolysaccharides of the cornea. The isolated mucopolysaccharide fractions used here are, of course, not identical with their state in situ, since such factors as hydration and linkages with the protein components of the tissue have been changed.

In an attempt to approximate more normal conditions, a suspension of finely minced fresh rabbit cornea was injected subconjunctivally into a group of three rabbits prepared as previously described, but the treatment was too irritating to the eyes for observations to be made.

One further attempt was made at whole

* Obtained from Dr. H. J. Anderson of the Cudahy Laboratories, Omaha, Nebraska.

tissue treatment, following the observations of Clark and Clark[9] in their studies on newly formed cartilage in the rabbit ear which showed a gradual retreat of blood vessels, previously present, before the advancing and differentiating area of regenerating cartilage. One to two square-mm. pieces of cartilage from the sternum of a young rabbit were implanted subconjunctivally into a group of three rabbits prepared as previously described, and observations were made over a period of four weeks. Here, again, there were no significant differences between the control and the treated eyes.

Summary

The subconjunctival injection of the isolated mucopolysaccharide fractions of bovine cornea had no effect on vascularization of the rabbit cornea caused by alkali and acid burns. Injection of minced fresh rabbit cornea, as well as the subconjunctival implantation of rabbit sternum cartilage, had no demonstrable effect.

210 East 64th Street (21).

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THE EFFECT OF VASODILATATING DRUGS ON CHOROIDAL CIRCULATION*

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The present experiments were carried out to investigate the effect of vasodilator drugs[1-18] on the choroidal circulation. The studies were confined to experiments in vivo.

Historical survey

The study of the drugs to be mentioned was inspired because, on reviewing the ophthalmic therapeutic literature, one or the other drug has been employed with reported success in pathologic afflictions of a vascular nature. Also, some of the workers studied the effect of some drugs on the choroidal circulation by indirect methods.

Ashton, Ward, and Serpell, in their study of the effect of oxygen in developing retinal vessels, also studied the effect on choroidal circulation in newborn kittens and rabbits. The method employed by them was the indirect method of observation.[4]

Anderson and Vallotton[6] reported the beneficial use of retrobulbar Priscoline in cases of occlusion of the central artery of the retina.

Viallefont[1] employed with beneficial results cobra venom as a vasodilator in cases of retinal vasoconstriction.

Gifford and Marquardt[2] reported vasodilatory effects with the injection of Papava-

* From the Department of Research, Wills Eye Hospital. This investigation was supported in part by a research grant from the National Society for the Prevention of Blindness.

rine hydrochloride (0.25 gm.) in cases of angiospastic retinopathy. They also reported beneficial vasodilatory effects with injections of typhoid vaccine in retinal vein thrombosis[16] and in cases of central angiospastic retinopathy.[2] The dosage employed by them ranged from 10 to 25 million bacilli.[16]

Tikhomirov,[3] in 1932, reported beneficial results after the administration of retrobulbar atropine in cases of angiospasm of the retina.

Anderson and Vallotton reported vasodilatory effects after retrobulbar injection of two-percent Novocain with hyaluronidase in cases of occlusion of the central retinal artery.

Duggan employed sodium nitrite as an ocular vasodilator in acute retrobulbar neuritis[10-12] and acute exudative choroiditis.[11-12] Esterman[13] reported the beneficial use of sodium nitrite in cases of acute spastic closure of the central retinal artery or one of its branches.

Sodium nitrite has also been employed in the form of Nitrosclerin as a general ocular vasodilator in various pathologic afflictions by Henry[14] and many others.[8-15]

Duff et al.[17] reported the vasodilatory effect of adenosine triphosphate when injected intrabrachially. It was pointed out by them that the blood vessels of the same hand and forearm were mainly affected. The drug had no effect on the blood vessels of the other parts of the body. The dosage employed ranged from 1.0 to 2,000 μg./min. and diluted in the ratio 64 μg./4.0 cc. of saline. The magnesium salt was reportedly a better vasodilator than the sodium salt of adenosine triphosphate.

Holten et al.[18] also reported vasodilatory effects of adenosine triphosphate.

Technique

The technique employed was the direct observation of choroidal circulation as described by Leopold, Vogel, and Wudka.[5]

The rabbit (albino) was anesthetized by intravenous pentobarbital sodium (Nembutal). Silk suture (4-0) was passed in the upper lid (near the lid margin) and the eye proptosed. The conjunctiva was dissected free of the muscles and the sclera. Another silk suture, preferably 4-0 silk, was passed (running suture) through the limbal conjunctiva to hold the eye in a desired position. The muscles were dissected free of the sclera and all the episcleral tissue dissected free of the sclera. The sclera was allowed to dehydrate. Bright focal illumination is obtained by the Barkan lamp or Nicholas lamp through the pupil. The large choroidal vessels and not the choriocapillaris were observed through a binocular stereoscopic microscope with magnification of ×10, ×40, and ×75.

The long postciliary artery is brought into view and its diameter is measured by a micrometer scale inserted in one of the oculars.

Experimental results

The following drugs were employed to study their pharmacologic actions on the large choroidal vessels. This method does not permit visualization of the choriocapillaris. The drugs were used by various routes. (See Table 1.)

Conclusions

1. Oxygen

Oxygen, 70 to 80 percent strength, when administered to newborn, one-day-old, one-week-old, and 10-day-old rabbits for a period of one hour did not affect the caliber of choroidal vessels.

2. Priscoline hydrochloride

Topical application of Priscoline hydrochloride (2.5 percent) had no effect on choroidal vessels. Retrobulbar injection in dosages varying from 25 to 50 mg. and intravenous and intra-arterial injections of 25 to 75 mg. had no effect on the choroidal vessels up to a period of one hour after administration.

TABLE 1

EXPERIMENTAL RESULTS*

Drug	Route of Administration	Dosage/Rabbit	Age	No. of Animals Studied	Effect on Choroidal Vessels
1. Oxygen					
A.	Inhalation for 45 minutes	70–80%	Newborn	6	No effect
B.	Inhalation for 45 minutes	70–80%	7 days	6	No effect
C.	Inhalation for 45 minutes	80%	10 days	6	No effect
2. Priscoline hydrochloride					
A.	Topical application	2.5%	Adult	6	No effect
B.	Retrobulbar injection	25–50 mg.	Adult	6	No effect
C.	Intravenous injection	25–75 mg.	Adult	6	No effect
D.	Intraarterial injection	25–75 mg.	Adult	6	No effect
3. Cobra venom solution (H. W and D) & Co.					
A.	Subcutaneous injection	1.0 cc.	Adult	6	No effect
B.	Intramuscular	0.50–1 cc.	Adult	6	No effect
4. Papavarine					
A.	Subcutaneous	0.25 gm.	Adult	4	No effect
B.	Intramuscular	0.25–0.5 gm.	Adult	6	No effect
C.	Intravenous	0.25–0.5 gm.	Adult	4	No effect
5. Typhoid vaccine	Intravenous	10–50 million bacilli	Adult	6	No effect
6. Atropine	Retrobulbar	1.0%–1.0 cc.	Adult	6	No effect
7. Novocaine C̄ epinephrine	Retrobulbar	0.50–2.0 cc., novocaine 2%, epinephrine 1:50,000	Adult	18	No effect
8. Procaine C̄ Wydase					
A.	Retrobulbar	1.0–2.0 cc., 1.0% Procaine C̄ 6.0 TRU units of Wydase to each cc.	Adult	12	No effect
B.	Retrobulbar	1.0–2.0 cc. of 2.0% Procaine C̄ 6.0 TRU units of Wydase to each cc.	Adult	12	No effect
C.	Retrobulbar	0.50–2.0 cc. of 4.0% Procaine C̄ 6.0 TRU units of Wydase to each cc. of Procaine	Adult	16	No effect
9. Sodium nitrite "Nitro comp" having the following composition: Sodium nitrite 100 mg. Sodium phosphate 3.6 mg. Dipotassium phosphate 2.0 mg. Sodium chloride in ampul water 6.0 mg.	Intravenous	75 mg.–200 mg.	Adult	6	No effect
10. Adenosine triphosphate†	Intraarterial	1000 μg–2000 μg	Adult	4	No effect

* All animals were observed up to a period of one hour after administration of the drug.
† The drug was diluted in 10 cc. of sterile saline and injected in the left carotid artery very slowly. The choroidal vessels of the left eye were observed.

3. Cobra venom

Subcutaneous and intramuscular injections of 0.5 to 1.0 cc. of cobra venom had no effect on the choroidal vessels up to a period of one hour after administration.

4. Papavarine

Papavarine (0.25 to 0.5 gm.) when given subcutaneously, intramuscularly, and intravenously had no effect on the choroidal vessels up to a period of one hour after administration.

5. Typhoid vaccine

Up to 50 million bacilli when given intravenously fail to produce any effect on the choroidal circulation.

6. Atropine sulfate

One-percent solution of retrobulbar injection did not indicate any change in the caliber of choroidal vessels.

7. Novocain with epinephrine

A dose of 0.5 to 2.0 cc. of two-percent so-

lution of Novocain when given by the retrobulbar route did not affect the caliber of choroidal vessels.

8. Procaine with Wydase

Strengths varying from one to four percent of procaine in varying amounts with six TRU units of Wydase to a cc. were given by retrobulbar route and failed to indicate any change in the caliber of the choroidal vessels.

9. Sodium nitrite

Intravenous injection of 75 to 200 mg. did not affect the caliber of choroidal vessels.

10. Adenosine triphosphate

Adenosine triphosphate (1,000 to 2,000 µg.) when injected in the left carotid failed to affect the choroidal vascular caliber in the left eye.

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PATRON GODS OF VISION

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When primitive man found the forces of nature difficult and unco-operative, he created gods whose function it was to preside over the uncertainties of his life and to whom he could appeal. There were gods for birth and death, hunting and agriculture, love and war, fertility and health. Some of these gods grew in prestige, married, acquired families, indulged in adventures marital and sexual, and became associated with numerous phases of human activity. With time and the merging and submerging of peoples, gods grew old, passed into retirement, and died, or several gods were combined to form a new one with multiple responsibilities.

Demons and evil spirits, and their human instruments, such as witches and wizards, were usually considered the cause of all disease, though a god might send disease as a punishment when angered by his people. Jehovah permitted the devil to cover Job with boils as a demonstration of the unfortunate man's faith. On such principles the only cure of disease was to invoke the aid of the benevolent gods, to propitiate and exorcise the evil gods and spirits, or both. Religion and magic struck directly at the assumed cause of disease. Treatment of the patient through drugs, diet, and hygiene could only alleviate uncomfortable symptoms and was of secondary importance. It is not an uncommon attitude in civilized society today. On occasion the nastiness of the medicines and the violence of the treatment were intended to make the human body an unpleasant habitation for the demon in possession.

No doubt any god who showed an interest in human welfare might receive an appeal for the preservation of health and the cure of disease. A few gods in each ancient culture became especially popular for their ability to cure the sick, and some specialists appeared who cared for particular diseases or parts of the body. Over 4,000 years ago the Sumerian mother of gods, Ninhursag, produced separate gods to treat pains in the hip, teeth, mouth, side, and ribs. There were other gods for other pains mentioned on the same cuneiform tablet from which these were deciphered but a complete translation could not be made.[1] It is not too unlikely that such an important organ as the eye also had a patron deity among the Sumerians.

The Assyrian god, Ea, was credited with supreme wisdom, general beneficence, power over evil spirits, and the creation of healing springs. Since Ea qualified so well for the role of healer, the sick naturally turned to him. His priest-physicians, dressed in robes resembling fish skins, responded with sacrifices, prayers, incantations, and a ritual employing water and oil.[2] For the afflicted simple remedies were prescribed to relieve discomfort until the god cured the disease. An Assyrian cuneiform tablet from the seventh century, B.C., translated by R. C. Thompson,[3] gives a prayer to Ea for the curing of a painful eye:

> In heaven the wind blew and brought blindness to the eye of the man: from the distant heavens the wind blew and brought blindness to the eye of the man. Unto the sick eye it brought blindness; of this man his eye is troubled, his eye is pained. The man weepeth grieviously for himself.
>
> Of this man, his sickness Ea hath espied and (said), "Take pounded roses, perform the Charm of the Deep, and bind the eye of the man." When Ea toucheth the eye of the man with his holy hand, let the wind which hath brought woe to the eye of the man go forth.

None of the gods of Egypt was particularly distinguished for curing the sick. Casting out the demons of disease was a minor function for them all. Horus, who had once suffered the loss of an eye, might be expected to be sympathetic, especially in cases of eye disease. The great sun god, Ra or

Atum, whose splendor was rarely dimmed by rain in the land of Egypt, certainly had the power if he could be persuaded. Osiris, as god of the dead, might grant a stay of execution if properly approached.

A prayer to these gods was combined with medication, in the medical Papyrus Ebers[4] of 1550, B.C., for the treatment of cataract. The green stone malachite, honey, and rush seeds were to be pounded together and applied to the eye, while an appeal was made for the assistance of "the discharge from Horus' eye," the "secretion from Atum's eye," and the "fluid that has come out of Osiris." This remedy, wrote the unknown author, was "really excellent." In fairness to the ancient Egyptian physicians it must be admitted that they seemed to place more emphasis on medication and less on prayers and incantations than their colleagues elsewhere. It was an advance not shared by the general public. The people preferred to place each part of the body under the protection of a special deity. The goddess Hathor was the protector of the eyes, as Osiris was the protector of the muscles.[5]

The posthumous career of Imhotep illustrates a theory advanced by Euhemerus of Sicily (fourth century, B.C.), who taught that all gods were merely kings and heroes deified by the people after death. In life Imhotep was the vizier of the Pharaoh Zoser, first king of the third dynasty who began his reign about 2980, B.C. Imhotep became famous as statesman, architect, and physician and, after his death, was elevated to the rank of patron saint or demigod of medicine. By 500, B.C., he had attained full divinity and consorted with the most venerable gods of Egypt.

When the sick came to the temples of Imhotep they were received by priests who described miraculous cures and recited prayers and incantations. With credulous minds prepared in this way patients slept in the temple, where they were visited by Imhotep in a dream, or by a priest in the guise of the divine healer. By morning the disease was cured, or at least instructions had been given for future treatment. This process is known as incubation or temple sleep. Grateful patients presented the temple with models of the diseased organ made of wood, blue glazed ware, or bronze, depending on their means, and these served as testimonials to impress future patients. When eye disease was cured a model eye was given to the temple.[6]

In the galleries adjacent to the temple of Asclepius the Greeks also practiced incubation, though among the more happy Greeks there was no organized priesthood but only temple attendants appointed by the government. To Homer, Asclepius was a highly successful general practitioner whose two sons went off to the Trojan war as army surgeons, but in time Asclepius became a hero and patron saint of medicine, and finally a god and the son of Apollo.

The earliest description of Greek incubation was given by Aristophanes[7] (448-380, B.C.) in his comedy of *Plutus*, in which Plutus, a personification of riches, recovers his eyesight by incubating at the Athenian temple of Asclepius and becomes better equipped to manage the distribution of wealth.

According to Pausanias[8] (second century, A.D.), the Asclepian temple at Naupactus was built by a grateful patient who had been blind. Asclepius was said to have instructed the poetess Anyte in a dream to take a letter, which she found beside her on waking, to the blind Phalysius. Long unable to read anything, Phalysius still, from a respect for divinity, made the attempt, and his vision was immediately restored. Following the instructions in the letter, he gave Anyte 2,000 gold staters, estimated at $9,500 in 1925, and, in addition, built a temple for Asclepius.

There were over 300 such temples in Greece and its dependencies, and in each one model limbs and organs accumulated, presented as exvotos by a constant stream of

patients. Among those at Athens were a silver heart, gilded legs, and gold eyes. Tablets were erected by those who had been cured, describing the circumstances, and at Epidaurus, the most important center of the cult of Asclepius, Pausanias saw six pillars covered with these testimonials. Two of the pillars have been excavated and, of 43 inscriptions remaining, eight refer to the cure of eye disease.[9] One of them seeks to convince by telling the story of the conversion of a sceptic.

> Ambrosia of Athens, blind of one eye. She came a suppliant to the god, and going round the temple ridiculed some of the inscriptions, saying it was incredible and impossible that the lame and the blind should be cured by seeing a dream-vision only. But having slept she saw a vision; the god seemed to stand by her and say that he would heal her, but would demand as payment a silver pig to be set up in the temple as a memorial of her stupidity. Having thus spoken, he opened her diseased eye and poured medicine on it, and when it was day she departed cured.

Another tells of the restoration of an eye that was entirely missing.

> A man once came who was so blind that one of his eyes had only the lids left, and seemed an empty space. Some persons in the temple ridiculed his simplicity, to hope for recovery when he had only an eye socket and no eye. He slept and had a vision; it seemed that the god prepared a medicine, separated his eyelids and poured it in. At daybreak he departed, seeing with both eyes.[9]

Because of an outbreak of disease in 291, B.C., the Romans sent an embassy to Epidaurus in order to obtain some symbolic representation of Asclepius for their stricken city. When a snake sacred to the god boarded their vessel the ambassadors decided they had what they needed and returned to Rome. There the snake determined the site of the new temple by leaving the boat and swimming to the island in the Tiber now called the island of St. Bartolomeo.[11]

To the temples of the Roman Aesculapius millions of exvotos were presented through the centuries. The street and bridge leading to the temple at Rome were lined with shops selling models of eyes and other organs in painted terra cotta, wax, plaster, bronze, silver, gold, and marble. Each temple had one or more rooms for the exhibition of exvotos hanging from nails or resting on shelves. When space became crowded, the cheaper exvotos were buried in pits, though at Rome they were simply thrown into the Tiber where many have been recovered during modern building operations. Similar exvotos were found buried at Rome beneath the ancient sites of the temples of Hercules, Juno, Diana Nemorensis, and Minerva Medica.[12]

As at Epidaurus, tablets were displayed recounting miraculous cures. Only a few from Rome, of doubtful authenticity, are available, but in these eye disease is prominent.

> In those days the oracle said to Caius the Blind that he should come to the sacred altar and kneel in front of it, then touch it on the left side, and instantly apply his hand to his eyes. Having obeyed, he recovered his eyesight at once, amidst the applause of the assembled multitude.[9]

Not everyone in the educated minority subscribed to this theory and practice of divine medicine. In the fifth century, B.C., the Greek physician Hippocrates[14] sought physical causes for disease and, in discussing epilepsy, wrote: "It is thus with regard to the disease called Sacred: it appears to me to be nowise more divine nor more sacred than other diseases, but has a natural cause from which it originates like other affections."

The Roman lawyer, Cicero[15] (106-43, B.C.) wrote: "How can it be reasonable for an invalid to apply for relief to an interpreter of dreams rather than to a physician? Can Aesculapius or Serapis, by a dream, best prescribe to us the way to obtain a cure for weak health? . . . Unfortunately the science of medicine cannot be learnt in dreams."

"I should have to be pretty far gone," said Lucian of Samosata[16] in the second century, A.D., "before I could admit that external things, which have no communication with the internal cause of disease, are going

to work by means of incantations and stuff."

Yet there are good reasons for the ancient popularity and continued existence of healing sanctuaries. Neurotic illness may indeed disappear overnight. Many diseases are self-limited and recovery is promoted by faith in supernatural assistance. Failures and relapses are not recorded, for the testimonial tablets were undoubtedly written with the guidance and hung with the permission of priests and temple attendants. The more remarkable cases, like that of the man who received an eye for his empty socket at Epidaurus, are products of the imagination inspired by abysmal ignorance or a deliberate intention to deceive the credulous.

1913 Spruce Street (3).

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Ophthalmic Miniature

> Then to several places to buy a hat, and books, and neckcloths, and several errands I did before I got home. And among others, bought me two new pair of spectacles of Turlington, who, it seems, is famous for them; and his daughter, he being out of the way, do advise me two very young sights, as that will help me most, and promises me great ease from them, and I will try them.
>
> *Pepys' Diary*, October 18, 1667.

THE TRANSMISSION OF LIGHT*

THROUGH THE OCULAR MEDIA OF THE RABBIT EYE

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INTRODUCTION

During a study of retinal damage in rabbits by thermal radiation, it became necessary to know the transmission of different wavelengths through the optic media in order to obtain threshold values of the thermal dose required to produce irreversible lesions. A review of the literature disclosed surprisingly little data which could be used. Roggenbau and Wetthauer[1] reported data on cattle eyes, while Ludvigh and McCarthy[2] measured transmission through four human eyes with normal optic media but with other pathologic changes. Kinsey[3] more recently has measured the transmission of ultraviolet through the rabbit eye but unfortunately his results extend only to 500 mμ. In what follows we wish to report transmission studies on nine freshly enucleated rabbit eyes in the wavelength range 380 to 1,400 mμ.

APPARATUS

Figure 1 is a schematic diagram of the apparatus used in our studies. A standard Beckman DU spectrophotometer serves as a monochromatic light source. The photocell box and cell carriage are replaced by a brass box containing a collimating lens, a 90 cycle per sec. light chopper, mirror, limiting aperture, and the eye mount. A 0.5-mm. diameter aperture placed 1.0 mm. beyond the adjustable slit of the spectrophotometer serves as a point source of light for the collimating lens. The eye is mounted within a plastic box, the cornea resting downward on a circular aperture matching the curvature of the globe.

A brass plate equipped with four needle points serves as a top plate; the needle points maintain the eye in a fixed position by contacting the sclera of the posterior pole at four points around a central hole which is trephined through the globe to the vitreous. This method effectively immobilizes the eye without damage or deformation in the desired position which is determined by viewing an image of the aperture on a ground glass placed behind the eye.

The eye mount is fastened to a polystyrene plate which can be slid out of the beam to allow a comparison with light which has not been transmitted through the eye.

Photocells were found to be unsuitable as detectors because their sensitivity varies with the area illuminated and the angle of incidence or divergence. In our earlier experiments using S-1 and S-4 vacuum photocells, we frequently obtained transmissions greater than 100 percent, despite our best efforts to keep the area of illumination and angle of incidence constant when the eye was moved in or out of the beam.

After trying several types of detectors, we chose the Kodak Ektron lead sulfide cell. This is a photosensitive semiconductor[4] consisting of a deposit of lead sulfide on a cover slip of glass with gold electrodes deposited on opposite ends. The sensitive area is a square, 1.0 by 1.0 cm. The Ektron cell has a useful range from 240 to 3,000 mμ with a peak sensitivity around 2,000 mμ. The sensitivity is independent of the area of

* From the Departments of Biophysics and Ophthalmology, Titmus Ophthalmological Research Laboratory, Medical College of Virginia. This research was supported by the U. S. Air Force under contract No. AF-18(600)-1272, monitored by the USAF School of Aviation Medicine, Randolph Field, Texas.

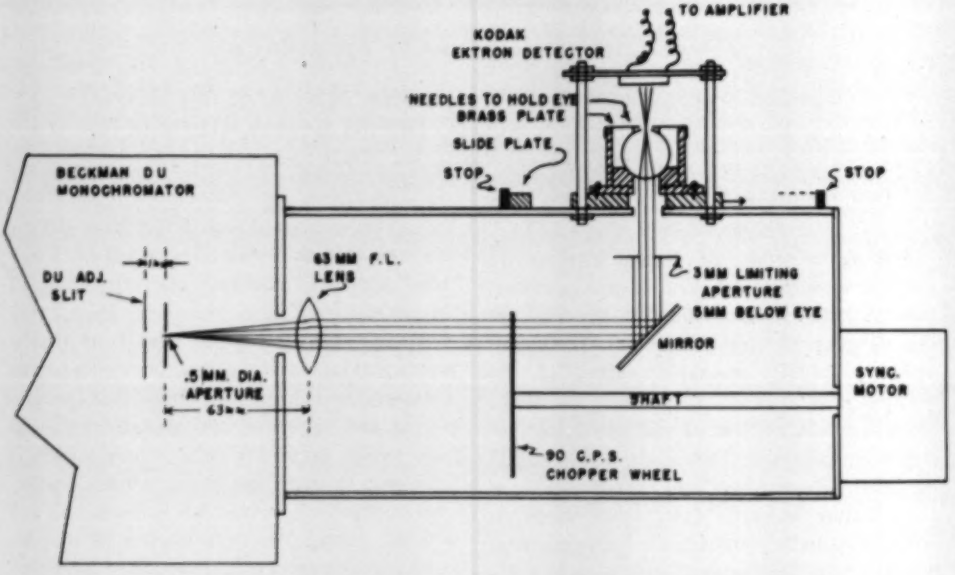

Fig. 1 (Wiesinger, et al.). Schematic diagram of apparatus for eye transmission studies.

illumination, so long as the flux is constant, and also independent of the location of the illuminated spot on the sensitive surface.

The Ektron detector, in series with a 1.8 megohm resistor, is polarized by a 90-volt battery. The a.c. signal appearing across the load resistor is capacity coupled into a two stage, tuned amplifier. Sharp tuning at 90 cycles per second is obtained with parallel T feedback networks from plate to grid of each stage. The amplifier response is down three decibels at ± three cycles per second off resonance, and has a voltage gain of approximately 65; it is linear for inputs up to 50 millivolts. A Ballantine model-300 vacuum tube voltmeter is used to measure amplifier output.

Experimental procedure

The eyes of mature Chinchilla gray and New Zealand black rabbits, ranging in weight from 3.0 to 4.2 kg., were used. All eyes were carefully examined and only those exhibiting ophthalmoscopically normal media were selected. Refractive error and corneal radius were measured on all eyes but found to have no significance insofar as transmission coefficients were concerned. After intraperitoneal sodium-Nembutal anesthesia (60 mg./kg. body weight) the eyes were enucleated in typical fashion.

The enucleated eye was placed immediately in physiologic saline solution and the sclera painstakingly freed from conjunctival tissue, muscle, fat, and clotted blood. Resting the anterior segment in saline solution, a hole, 5.0 mm. in diameter, was cut by means of a Storz trephine through the sclera to the vitreous at the posterior pole just below the optic nerve. The eye was mounted in the plastic cup, adjusted in position by observing the image of the spectrophotometer aperture on a ground glass plate, and immobilized in the correct position by the needles of the top plate. Since post-mortem constriction invariably fixed the pupillary diameter at 3.5 to 4.0 mm., no difficulty was experienced in passing the full beam of light defined by the 3.0-mm. diameter limiting aperture through the eye

TABLE 1

TRANSMISSION DATA FOR NINE RABBIT EYES

Wavelength (in mμ)	Mean Percent Transmission for Nine Eyes	Standard Deviation	Coefficient of Variation (%)	Wavelength (in mμ)	Mean Percent Transmission for Nine Eyes	Standard Deviation	Coefficient of Variation (%)
380				780	93.2	1.15	1.2
390	46.5	4.50	9.7	800	93.2	1.43	1.5
400	61.0	3.02	5.0	820	93.8	1.52	1.6
410	71.4	3.67	5.1	840	92.4	1.35	1.5
420	75.4	3.01	4.0	860	91.5	1.08	1.2
430	79.1	3.14	4.0	880	90.4	1.64	1.8
440	82.9	2.32	2.8	900	88.2	0.84	1.0
450	85.2	2.16	2.5	920	86.3	1.45	1.7
460	87.9	3.06	3.5	940	78.1	1.19	1.5
470	88.7	2.68	3.0	960	61.5	2.94	4.8
480	90.5	2.45	2.7	980	51.9	1.00	1.9
490	90.8	2.06	2.3	1000	57.5	6.30	11.0
500	92.4	1.80	1.9	1020	64.3	5.81	9.0
520	92.9	2.56	2.8	1040	71.4	1.11	1.6
540	93.5	2.15	2.3	1060	77.7	0.95	1.2
560	94.4	1.73	1.8	1080	79.8	1.10	1.4
580	93.5	0.87	0.9	1100	76.5	1.20	1.6
600	93.5	1.57	1.7	1120	70.3	1.27	1.8
620	93.5	0.92	1.0	1140	53.5	2.46	4.6
640	94.4	1.07	1.1	1160	22.8	3.10	13.6
660	94.7	1.25	1.3	1180	16.8	0.44	2.6
680	95.2	1.32	1.4	1200	15.6	0.69	4.4
700	94.6	1.63	1.7	1250	20.9	0.91	4.4
720	94.4	1.24	1.3	1300	18.1	0.88	4.9
740	92.8	1.44	1.6	1350	2.4	0.22	9.2
760	92.6	1.95	2.1				

and onto the detector. It was unnecessary to adopt any special precautions for maintaining the corneal surface in a moist condition since all measurements were completed before any signs of drying became evident.

Transmission measurements were begun not later than 15 minutes after enucleation and completed within 45 minutes thereafter. For each wavelength setting, the reading of the voltmeter was adjusted to a suitable value by varying the slit width of the spectrophotometer; the eye was then moved into the beam and the reduced reading recorded. This procedure was repeated every 10 mμ from 390 to 500 mμ, every 20 mμ for the interval 500 to 1,200, and every 50 mμ over the range 1,200 to 1,400. Transmission virtually ceased at 1,400 mμ. After careful examination of the cornea to make sure that the surface was moist and in optimum physiologic condition, the measurements were repeated at wavelengths of 400, 500, 600, and 700 mμ. These subsequent measurements agreed with the original to better than five percent in all cases.

RESULTS

Transmission data for nine enucleated rabbit eyes are given in Table 1 where the first column records the wavelength in mμ, the second column records the mean percentage transmission for nine eyes at each wavelength, the third column gives the standard deviation, and the fourth column gives the coefficient of variation. It will be noted that the maximum coefficient of variation observed was 14 percent at a wavelength of 1,160 mμ. Through most of the spectrum the coefficient of variation is less than five percent.

In Figure 2, the mean percentage transmission for nine eyes is plotted against wavelength in mμ, where for purposes of

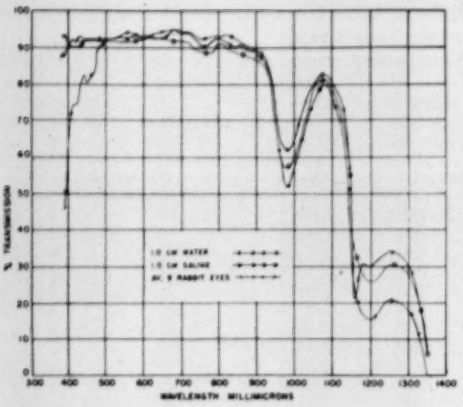

Fig. 2 (Wiesinger, et al.). Transmission curve for rabbit eyes.

comparison we have plotted also the percentage transmission through one cm. of distilled water and one cm. of physiologic saline solution. While the optic media of the rabbit eye has a thickness greater than one cm., it is obvious that the general form of the transmission curves are remarkably similar in the three cases. This recalls the observation of Aschkinass[5] who in spite of a lack of accurate data predicted that transmission through the eye was entirely similar to that through water. Our results agree reasonably well with those reported by Kinsey[3] over the wavelength range 380 to 500 mμ. It is planned to perform these measurements on a number of human eyes as the opportunity arises.

Summary

A technique to measure transmission through the optic media of freshly enucleated rabbit eyes which is thought to be free of major sources of error is described. A Beckman DU Spectrophotometer is used as a monochromatic light source. The Kodak Ektron cell is employed as a detector because of its useful range of 240 to 3,000 millimicrons and because the sensitivity is reasonably independent of area of illumination and angle of incidence.

A composite curve of nine rabbit eyes examined by this method is presented and plotted against transmission curves of one cm. water and one cm. saline to demonstrate their similarity above 500 mμ.

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Ophthalmic Miniature

"Congestive oedema" characterizes all acute and sudden changes in intraocular pressure, and hence blocking of the Angle is a constant and marked pathological feature in acute Glaucomas; while in the chronic noncongestive forms it is absent, and the Angle is found open.

Thomas Henderson,
Glaucoma. London, E. Arnold, 1910, p. 150.

TREATMENT OF SENILE MACULAR DEGENERATION*

By advancing the inferior oblique muscle

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Advancing the medial one half of the inferior oblique muscle insertion to the area of the short and long ciliary vessels of the eye, in an effort to establish a collateral blood supply to the choroid in the avascular area of the macula, may be of value in the treatment of senile macular degeneration. The highly vascular muscle tissue of the inferior oblique is two or three mm. from the macula and is readily available for transplantation to the area of the sclera where it is perforated by the posterior ciliary vessels.

A review of the literature offers little definitive treatment for senile degeneration of the macula. The use of the old and new vasodilators, iodides, B-complex, rutin, vitamin E, and the newer hypertensive drugs in the medical care of the patient's general vascular disease offers some assistance in delaying the loss of macular vision. The transplantation of placental tissue offers some benefit and anticoagulants may prove helpful.

The pathogenesis of macular degeneration is still in doubt but the consensus seems to be, as pointed out by Verhoeff and Grossman,[1] a disturbance in the choriocapillaris and local angiosclerosis of the choroid in the area of the macula. Brown[2] agreed with this opinion and felt the retinal vessels were secondarily involved, with the choroid the primary seat of the disease. Hartridge[3] showed histologically and subjectively that the macula does not have retinal vessels and is thus supplied primarily from the choroid. Sun[4] reported a patient with disciform degeneration in one eye, and circinate retinopathy in the opposite eye, illustrating diseases of the separate vascular systems of the eye.

Gifford[15] suggested sectioning the oblique muscles for whatever influence this might have in aiding the vortex veins to improve the macular circulation. Grossman and Pischel believe that many of the favorable results in detachment surgery may be due to improved circulation to the eye brought about by the surgery establishing new vessels from the retrobulbar space. Macular vision is impaired if the retina has been detached for over one month, showing the importance of the choroidal supply to the macula.

It has been observed during the past three years that eight cases operated in the posterior segment of the eye for detachment of the retina, intraocular foreign bodies, Von Hippel's disease, and for transillumination for possible tumors, showed definite improvement in their lesions due to senile macular degeneration. Careful review of the vision and reports of macular disturbances preoperatively and postoperatively in patients with surgery of the posterior segment of the eye may prove valuable. It seems that more active treatment of macular degenerative diseases can be achieved by surgical means.

It was felt that this matter was worth investigating and experimental work was done on Rhesus monkeys. The surgery was done under deep Nembutal anesthesia with reasonable asepsis, and a single dose of penicillin was given postoperatively to prevent infection. Rhesus monkeys 1 and 2 were operated on April 3, 1955, with the following procedures:

O.D., Monkey 1. The lateral rectus tendon was cut between sutures and the sutures used for traction. The medial one half of the right

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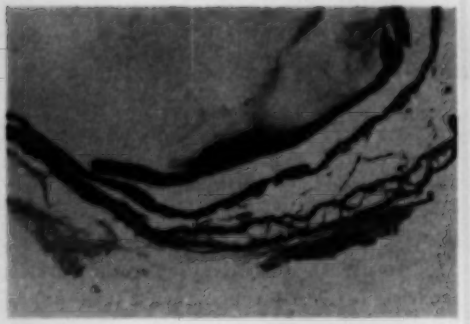

Fig. 1 (Burnside). O.D., Monkey 1. *Simple advancement.* Posterior segment of the eye, showing numerous capillary type blood vessels with moderate fibroblastic infiltration.

inferior oblique muscle was advanced two to three mm. over the macula and sutured with 6-0 mild chromic suture.

O.S., Monkey 1. The same procedure with the exception that four superficial cautery applications were made to the sclera.

O.D., Monkey 2. The inferior oblique was advanced as described over a single radial two mm. incision through the sclera.

O.S., Monkey 2. The same procedure as for OD. was carried out except that an effort was made to place the muscle intrasclerally beneath the choroid but with a tear

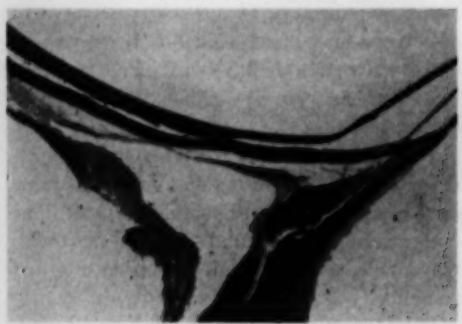

Fig. 2 (Burnside). O.S., Monkey 1. *Advancement with cauterization.* The infiltration of blood vessels in the area of the cauterization beneath the advanced muscle presents a larger size ratio in the blood vessels. There are small areas of necrosis of the superficial sclera and disorganization of the retinal cells in the macular area.

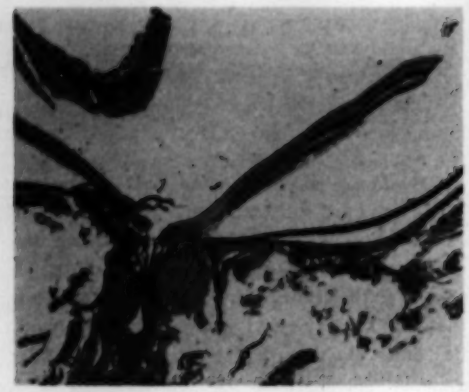

Fig. 3 (Burnside). O.D., Monkey 2. *Advancement with posterior sclerotomy.* The blood vessel pattern is similar to that seen in the eye of Monkey 1 but with a few pigment cells in the extrascleral adhesions. The choroidal vessels are engorged and the entire layer is thickened.

in choroid and with a moderate loss of vitreous.

Monkey 1 and Monkey 2 had no ocular complications and did well postoperatively. Monkey 2 developed pneumonia on the 65th postoperative day and was killed. Monkey 1 was killed on the 84th postoperative day.

Monkey 3 was operated, left eye only, on July 12, 1955, with a transplant of the left inferior oblique over the macula; while under deep anesthesia with the pupils widely dilated, the macular areas of both eyes were

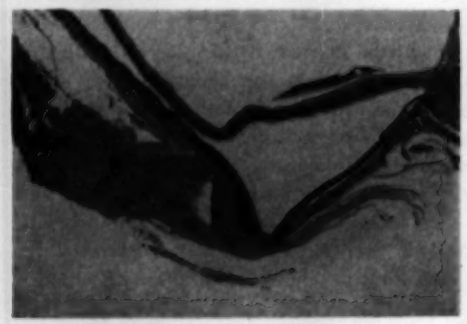

Fig. 4 (Burnside). O.S., Monkey 2. *Intraocular implant.* There is extensive granulation type of tissue about the area of the intraocular transplant, with marked migration of the pigmented choroidal cells into the muscle fibers and granulation tissue.

exposed to direct sunlight for four minutes. This animal was killed on the 60th postoperative day.

Pathologic report

The clinical pathologic report on the six eyes follows:

O.D., Monkey 1, showed a normal fundus picture with no macular lesion pre- or postoperatively. Grossly the eye was normal except for dense adhesions on the sclera in the areas of the macula and lateral rectus, most marked in the macular area. Microscopically, the posterior segment of the eye showed numerous capillary vessels with moderate fibroblastic infiltrations.

O.S., Monkey 1. The fundus was normal preoperatively but postoperatively showed loss of normal reflex and color of the macula, with early pigmentary disturbances. Grossly, the posterior pole of the eye presented a very large area of adhesions. Microscopically, the blood vessels showed a larger size ratio. There were small areas of necrosis in the superficial sclera with some round-cell infiltration and disorganization of the retinal cells in the macula.

O.D., Monkey 2, presented a normal fundus pre- and postoperatively. Microscopically, the blood-vessel pattern was similar to the eye of Monkey 1 with the added feature of a few pigment cells in the extrascleral adhesions. The choroidal vessels were very engorged and the choroid layer was thickened.

Fig. 5 (Burnside). O.D., Monkey 3. *Sunburn without muscle advancement.* The eye was characterized by increased thickness of the choroid, round-cell infiltration into the choroid, and disturbance of the retinal pattern.

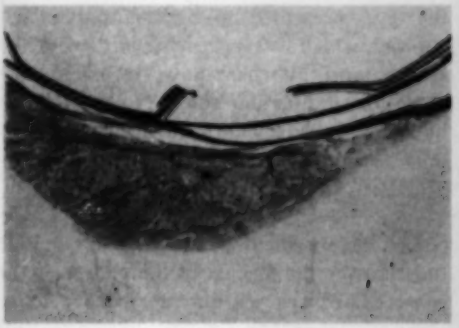

Fig. 6 (Burnside). O.S., Monkey 3. *Sunburn with advancement.* The posterior granulation is most marked in the macular area, with a surprisingly small amount of disturbance of the retina and choroid in view of the severe burn inflicted.

O.S., Monkey 2. The fundus was normal preoperatively but postoperatively presented a small flat detachment below the macula with a large choroidal tear immediately lateral to the macula. Grossly, the eye seemed smaller than O.D., 2, with very dense adhesion masses posteriorly. Microscopically, the eye presented extensive granulation of tissue about the area of sclera transplant, with marked migration of the pigmented choroidal cells into the muscle fibers and the granulation tissue.

O.D., Monkey 3. The fundus was normal preoperatively; postoperatively, it had a dense white area of the entire central retinal area immediately. At the end of the 60th postoperative day, the macular area was a dirty gray color, extending centrally over an area to include the disc. Histologically, the eye showed increased thickness of the choroid, round-cell infiltration of the choroid, and disturbance of the retinal pattern.

O.S., Monkey 3. The fundus was normal preoperatively. Postoperatively, it showed slight pallor immediately after exposure to the sun. At the 60th postoperative day it had a normal appearing fundus. Microscopically,

the eye presented a normal retina and choroid, which was surprising in view of the severe burn inflicted; however, there was posterior granulation similar to that shown by a simple transplant of the inferior oblique muscle without burn.

Summary

The advancement of the inferior oblique muscle over the macula in the region of the short ciliary arteries may establish a long-lasting collateral blood supply when a physiologic need is present. The patient with advanced arteriosclerosis should, if possible, have a posterior sclerotomy beneath the transplant for collateral circulation to the choroidal vessels plus anastomosis to the short ciliary arteries. The use of mild chromic sutures seems in itself to augment the number of vessels established in the site.

No untoward cosmetic defects, such as squint or ptosis, were noted in any of the eyes following surgery. The animal used in an effort to produce actual advanced macular degeneration by direct sunlight surprisingly did not show the expected reaction immediately after operation or after the two-month interval. Possibly the marked hyperemia produced by the operative procedure rapidly dissipated the heat and prevented extension of the damage to the retina. Could this help to minimize macular burns from atomic radiation? The anastomosis was most marked following infrared burns of the macular area, as would be expected on the basis of physiologic need.

Conclusion

Advancement of inferior oblique muscle with or without posterior sclerotomy may be a means of establishing collateral circulation to the choroid beneath the macula in presenile and senile degeneration. This is a relatively simple procedure without serious complications to the eye. Possibly, it should be done first on the poorer eye in individuals with less than 20/50 vision.

4105 Live Oak (1).

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NOTES, CASES, INSTRUMENTS

GLAUCOMA DEVELOPING IN A MORIBUND PATIENT*

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A patient was seen with primary bronchogenic carcinoma, with metastasis to the regional lymph nodes, liver, right kidney, both adrenals, brain, right sixth rib, and left choroid and ciliary body. A short time before death from generalized carcinomatosis, she developed a glaucoma in the right eye. After death, the eyes were obtained for examination. The findings in the eye with metastatic lesions were previously reported.[1] The case report in brief, as pertains to the glaucomatous eye, is as follows:

CASE REPORT

The patient, a 59-year-old woman, was seen with the complaint of a partial loss of the left temporal field of vision for four or five weeks. Her history revealed the occurrence of mild pain and lacrimation in that eye for a corresponding period of time. A chronic progressive cough had been present for 18 months, with occasional blood-streaked sputum. Bronchoscopy and examination of a coughed-up specimen had revealed the presence of a nonmalignant adenoma of the left upper bronchus. On the appearance of a solid-appearing detachment of the retina in the left eye and the history, a diagnosis was made of metastatic carcinoma of the choroid. Other metastases were suspected and proved to be present on autopsy three months later.

After two months' hospitalization, the patient complained of loss of vision in her uninvolved eye. On examination, the cornea was steamy and the eye was found to have a tension of 80 mm. Hg. (Schiøtz). Treatment was instituted with one percent pilocarpine, and in four days the tension had receded to 44 mm. Hg. The examination of the fundus was negative. Two-percent pilocarpine three times a day lowered the tension to 30 mm. Hg in five more days. The anterior chamber appeared to be normal in depth, and the patient was symptomatically cured insofar as this eye was concerned. She died one month and 12 days after the first symptoms of glaucoma.

During the three-month period of hospitalization, the patient was treated symptomatically. She was able to retain only small amounts of food or fluids, and was given five-percent dextrose solution intravenously, receiving up to two liters daily. Her general condition gradually became worse. Occasionally during hospitalization, she could retain some fluids by mouth. In these instances, her glucose administration was decreased according to the amount retained.

The eye was obtained at necropsy and examined by Dr. Michael J. Hogan of the Proctor Laboratories, University of California. The abstracted pathologic report is given with his permission.

PATHOLOGIC REPORT

Gross examination. The right eye measured 24 by 24 by 25 mm. It appeared normal.

Microscopic examination. The circumcorneal tissue, cornea, limbus, and sclera were normal. The temporal angle of the anterior chamber showed a very narrow synechia (fig. 1). The nasal angle was entirely open (fig. 2). The chamber had a normal depth. The iris stroma showed a moderate amount of chronic inflammation. There was moderate hyalinization of the ciliary processes. The stroma and muscularis of the ciliary body appeared normal. The choroid, retina, vitreous, and lens showed no abnormal findings.

* From the Medical Department, United States Air Force.

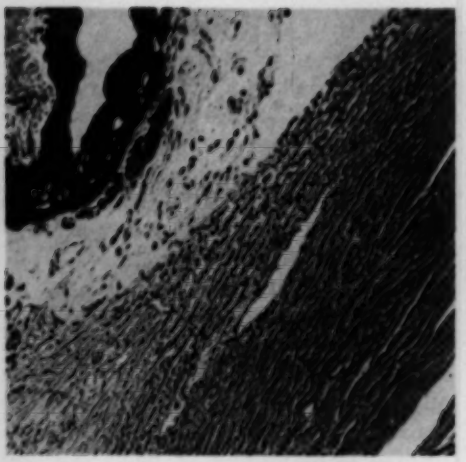

Fig. 1 (Maxwell). Showing peripheral anterior synechia.

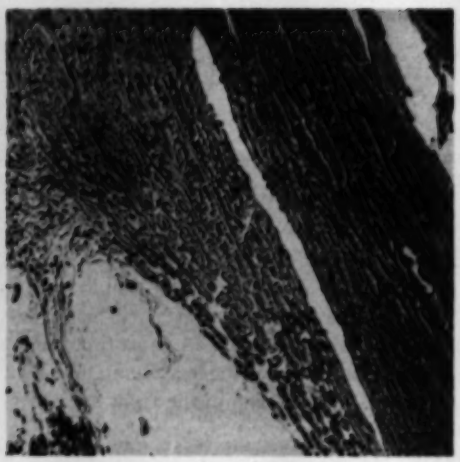

Fig. 2 (Maxwell). Showing open angle opposite Figure 1.

The optic disc showed normal vessels with no cupping.

Diagnosis. Synechia, peripheral anterior, iris, partial.

Comment

The glaucoma developing in the right eye of the patient was difficult to explain. It was of the simple (open-angle) type and occurred in a patient who was being treated with large amounts of intravenous fluid. The fundus had been examined carefully at least three times a week for over a month prior to the attack, since a search was being made for metastasis. The lens gave no evidence of swelling and apparently could not have pushed the iris forward closing the angle. Although Duke-Elder[2] believes the difference in osmotic pressure between the blood and aqueous humor to be of limited clinical significance, perhaps this was a factor in this case. The trabecular spaces were open, the angle was wide, the tension was high for only a short period of time, and yet, interestingly enough, one peripheral anterior synechia was formed.

Norton Air Force Base.

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THE MICROSCOPE IN OCULAR SURGERY

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Today, many of the ordinary maneuvers in ocular surgery need the use of a magnifying lens. In some cases, a great magnifying power is required. The corneal microscope may be used for minute and delicate procedures, such as the extraction of corneal foreign bodies, sutures points, and so forth. However, the use of the corneal microscope and slitlamp is limited because with these instruments the patient must be in a sitting position which is satisfactory for examination but not for intraocular surgery.

The Littmann-Zeiss surgical microscope permits use of high-power magnification—with the patient in a horizontal position. It

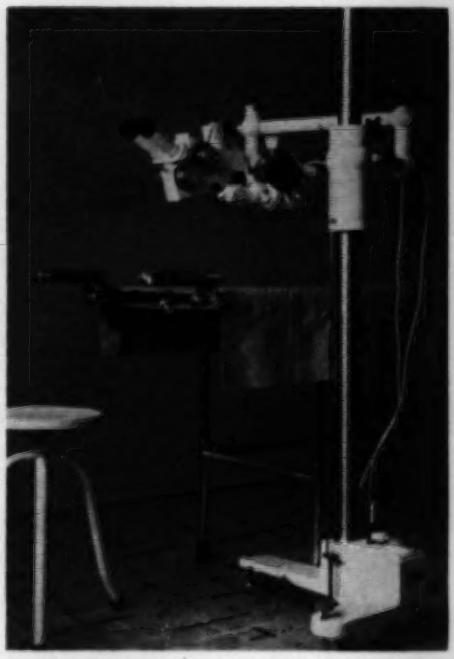

Fig. 1 (Barraquer). The Littmann surgical microscope with pedal focus control and the slitlamp on a special arm which permits adjustment to any position.

is particularly useful in extracting foreign bodies from the cornea and anterior chamber, for the rupture of the synechias, for the extraction of capsular debris, in dissecting the cornea in lamellar keratoplasty, in extraction of corneal and corneoscleral sutures, in estimating the depth of an incision, in determining the position of the hyaloid in aphakic eyes in which it is necessary to do a paracentesis of the anterior chamber, and so on.

Use of this instrument also permits visualization and elimination of those foreign bodies which constantly invade the operative field, such as cotton fibers and talcum powder—particles invisible to the naked eye that may enter the operative wound, and cause postoperative complications.

The surgical microscope, as it is constructed, presents no difficulty in exploratory procedures or for simple operations which can be done with one hand while the other hand adjusts the microscope to maintain correct focus and centering. However, in more complicated operations, difficulties arise: magnification is hard to adjust; there is false projection of the image; it is difficult to maintain the operative field in correct focus.

The difficulties arising from magnification or false projection are controlled with practice only. False projection is less of a problem when the objective is inclined so that the operative field seen through the microscope is the same as that observed on looking down without the microscope.

Displacement of the operative field can be avoided by good general akinesia; in special cases, the surgeon can make corrections by moving the patient's head, or an assistant can adjust the microscope as the surgeon directs, or the operative field can be centered by means of the light source of the instrument.

Focus changes should be made by the surgeon. The problem of doing this while working with both hands has been solved by construction of a foot control which adjusts the focus instantly on pressure with the right foot. By this pedal attachment it is possible to maintain the correct focus for each successive operative step. With this attachment, the observation field of the microscope does not change when it is in a vertical position but does change in an inclined position; however, displacement of the image in ocular surgery is not important, if one keeps in mind that focussing displacements almost never reach five mm.

The axial illumination which the microscope provides is excellent for eyes without photophobia or for well-anesthetized eyes; but for sensitive eyes, and especially for extraction of sutures, I have found a way to lower the intensity of the light.

Use of the slitlamp permits observation of the optical section. When the slit is completely open, lateral illumination, which has great advantage in some ocular interventions, is provided, for example, in anterior segment

surgery, the visibility is better with lateral than with axial illumination.

The slitlamp (Zeiss standard model) has been firmly attached to the microscope by means of a special arm which permits adjustment in any position. To the optical system I have added a 10-diopter concave lens for focussing at a distance of 15 cm. The focus of the slitlamp and microscope can be adjusted to the same plane before the operation. During the operation, the lamp and microscope will focus on the same plane and this plane can be controlled with the pedal attachment.

Adapting the slitlamp to the surgical inicroscope gave excellent results in lamellar keratoplasties because it was possible to visualize the thickness of the receiving bed. It is also useful in extraction of foreign bodies situated in the deeper layers of the cornea and for biomicroscopic observation of the anterior segment, fundus, and anterior-chamber angle in anesthetized children.

Apartado Aereo 11056.

TUBE FOR CORNEAL BATHS*

O. A. Capriotti, M.D.
Souderton, Pennsylvania

Although there are many tubes for corneal baths, most of them are designed principally for iontophoresis. The tube herein described was designed primarily for corneal baths. It is so constructed that it can be used even when the patient is in a sitting position.

The scleral ring measures 24 by 26 mm. and is cupped 10 mm. to clear the cornea. This makes a snug fit so that when used in the sitting position there is little, if any, leakage. The tube measures about 55 by 10 mm. and has a capacity of about six cm. of fluid. The opening of the tube is at the upper surface.

Fig. 1 (Capriotti). Tube for corneal baths.

Fig. 2 (Capriotti). The tube in use with the patient in a sitting position.

A sling of scotch tape fastened to the forehead can be used if the distal end drops while the patient is in a sitting position.

This device is simple to manage, thus making the corneal bath a feasible office procedure.

404 East Broad Street.

* This tube is manufactured by the R. O. Gulden Company, Philadelphia 20, Pennsylvania.

INCREASING IMAGE MAGNIFICATION*

IN RETINAL PHOTOGRAPHY

JACK H. PRINCE, F.R.M.S. (ENG.)
Columbus, Ohio

By increasing the camera tube length of the Zeiss-Nordensen retinal camera and using the auxiliary lenses incorporated in the instrument, a much larger picture of a small lesion can be obtained. In fact the optic nerve can be made to cover most of a 35-mm. frame without noticeable reduction in resolution.

The tube immediately in front of the camera body and behind the light deflection prism is detachable. If this is replaced with a tube six inches longer and the −0.75D. auxiliary lens is turned into position, an increase in magnification of ×2 is obtained. If the tube length is increased to eight inches and the −1.5D lens is used, then the magnification is even greater. The entrance pupil is changed by this procedure but it does not affect the picture unduly. The field stop is provided by the camera frame.

The mathematically ideal lengths of tube are probably a little longer than quoted here, but the focusing adjustment has sufficient latitude to compensate for the difference.

* From the Department of Ophthalmology, The Ohio State University.

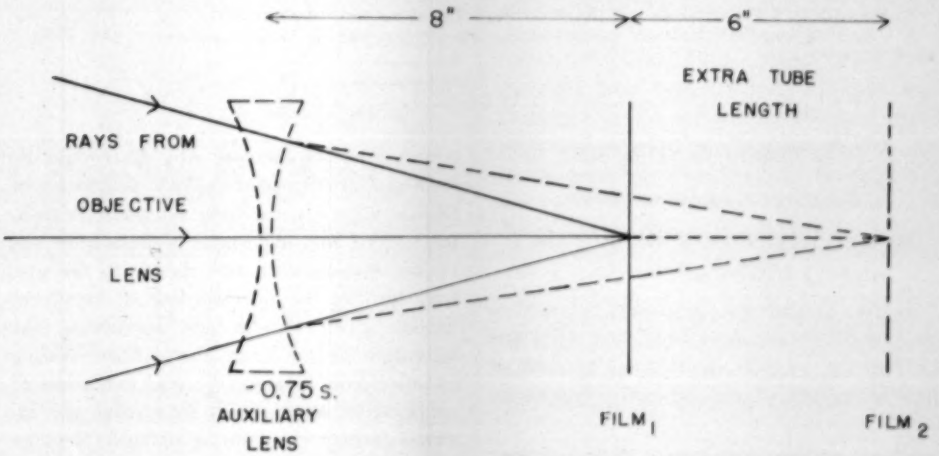

Fig. 1 (Prince). Diagram showing the simple alteration in the effective focal length of the camera objective to produce a larger image.

A TIMING UNIT FOR TONOGRAPHY*

S. I. ASKOVITZ, M.D.
Philadelphia, Pennsylvania

With the increasing interest in aqueous outflow determinations[1] during the past few years, efforts have been made to simplify clinical tonography so that the procedure may be readily applied by any ophthalmologist. The present paper describes a small timing unit designed toward this end.

For all practical purposes, it suffices in general for an assistant to write down the tonometric readings every 15 seconds[2] throughout the four- or five-minute total interval, thus eliminating the need for any expensive auxiliary recording equipment.[3] However, to note the tonometer values at regular intervals soon becomes monotonous for both tonographer and assistant, and it is easy to miss one or more readings, even with

* From the Ophthalmology Research Laboratory (Dr. I. H. Leopold, director), Albert Einstein Medical Center, Northern Division. This work was supported by a grant from the Weinstock Fund.

Fig. 1 (Askovitz). A timing unit for tonography.

a watch or stop-watch at hand.

It was therefore considered advisable to have a simple timer to serve as reminder. Most commercially available signal timers require resetting at the end of each interval and are therefore not too convenient. A simple arrangement designed specifically as an aid in manual tonographic recording is shown in Figure 1.

The basic mechanism consists of a precision timing motor which rotates a notched cam once each 15 seconds, providing a long cycle of 14 seconds alternating with a short cycle of one second. The cam acuates a microswitch which turns on a jewelled pilot light during the "long" period and a small buzzer for the "short" period.

The volume control permits adjusting the loudness of the buzzer or shutting it off altogether and leaving only the pilot light as timer. If desired, the whole assembly could be incorporated on the control panel of the voltage stabilizer and ground-testing unit[4] recommended for use with the electronic tonometer.

Other applications for this timing unit include its use as a fixation light for young children; the bulb's flashing off and on provides added stimulus for the child to keep his attention in the direction of the light. It may also be used as a reminder and timer for the repeated instillation of eyedrops, and so forth.

York and Tabor Road (41).

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HORNER'S SYNDROME*

FOLLOWING PHRENIC CRUSH

B. M. CHATTERJEE, F.R.C.S. (ENG.)
Calcutta, India

Horner's syndrome, originally referred to by Claude Bernard[1] in 1852, but described by Horner[2] in 1869, develops as a result of paretic lesions of the cervical sympathetic connections for the eye. Any destructive lesion—traumatic, inflammatory, neoplastic, or degenerative—in the long sympathetic pathway from the hypothalamus down to the upper thoracic cord and thence to the eye, may lead to the development of this syndrome. Even several odd conditions like laceration of the sympathetic fibers during tonsillectomy (Hammer[3]) and extension of suppurative otitis media to involve the internal carotid sympathetic plexus (Metzner and Wölffin[6]) have been reported.

The following is the report of an unusual

* From the Eye Department, Medical College, Calcutta. Presented before the Ophthalmological Society of Bengal on May 29, 1954.

TABLE 1

EFFECT OF DRUGS ON PUPIL IN LESIONS OF DIFFERENT SYMPATHETIC NEURONES

	Neurone III (Postganglionic)	Neurone II (Preganglionic)	Neurone I (Central)
Cocaine	−	−	++
Adrenalin	+++	−	−

+ Dilatation.
− No action.

case of Horner's syndrome developing after a phrenic crush in a patient suffering from pulmonary tuberculosis.

CASE REPORT

S. D. C., a man, aged 24 years, with an established case of pulmonary tuberculosis and undergoing treatment at the chest clinic of the Medical College Hospitals, Calcutta, complained that about a month ago, on the day following an operation for phrenic crush on the right side, his right upper eyelid was drooping.

Eye examination. Right eye. There was partial ptosis with narrowing of the palpebral fissure. The action of the levator was unimpaired. There was slight apparent enophthalmos. The movements of the eyeball were normal. There was no vasodilatation of the conjunctival vessels. Cornea and other ocular media were clear. The pupil was definitely smaller in size than that of the other side, the ratio being 2.5 mm.:4.0 mm. but the reactions of the pupil to convergence, accommodation, and light were normal. There was no depigmentation of the iris. The fundus picture and the intraocular pressure were normal. The vision was 6/6.

Left eye. Vision was 6/6 and there was no other abnormality.

On the instillation of cocaine hydrochloride (four percent) in each eye four times every 10 minutes, there was no action on the right pupil, but the left pupil dilated to the size of about 9.0 mm. A solution of adrenalin chloride (1:1,000) when dropped in each eye, every three minutes for three times, had very little effect on either pupil, even after a period of 45 minutes.

A skiagram of the chest showed tuberculous infiltrative lesions toward the base of the right lung. Other general and neurologic examination did not reveal any other pathologic abnormality except an operation scar on the posterior border of the lower part of the right sternomastoid. The condition was diagnosed as Horner's syndrome.

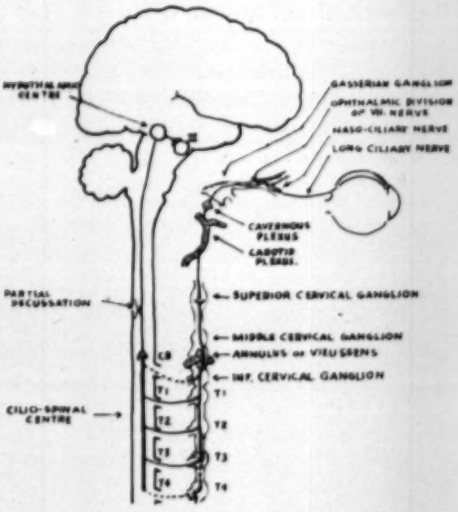

Fig. 1 (Chatterjee). Cervical sympathetic with pupillary connection. (After Duke-Elder.)

DISCUSSION

As shown in Figure 1, the long pathway of the cervical sympathetic system for the eye consists of three neurones. The first neurone extends from the sympathetic center in the hypothalamus to the ciliospinal center of Budge in the anterolateral tracts of the spinal cord at the level of the seventh and

eighth cervical and first thoracic segment. The second neurone leaves the cord by the anterior spinal roots mainly of T1, of T3 and sometimes of C8 also, passes through the inferior and middle cervical ganglion of the sympathetic, and extends up to the superior cervical ganglion along the cervical sympathetic chain. The third neurone originates from the superior cervical ganglion, enters the skull with the carotid plexus, passes through the cavernous plexus, and ultimately supplies the involuntary muscles of the eyelid and the dilator pupillae.

Localization of a lesion to any one of these neurones can be done according to the pharmacodynamic tests advanced by Foerster and Gagel[2] in 1932, as shown in Table 1. These tests showed that the affected pupil had no reaction either to cocaine or to epinephrine and so, according to Table 1, the lesion was located in the second neurone of the sympathetic. In the absence of any other obvious cause, it was concluded that the surgical interference necessary for the phrenic crush could somehow be responsible for such a lesion.

The phrenic nerve in the neck is usually approached from the posterior triangle along the posterior border of the lower part of the sternomastoid muscle. The nerve lies on the anterior surface of the scalenus anterior muscle, whereas the sympathetic chain lies in a much deeper plane behind the carotid sheath. Possible explanations for the injury to the sympathetic are probably:

a.[5] At the root of the neck each phrenic nerve is joined by a filament from the sympathetic and an attempt to pull out the phrenic nerve may drag on the sympathetic chain.

b.[5] The middle cervical ganglion of the sympathetic is connected to the inferior cervical ganglion by two or more cords, one of which forms a loop around the subclavian artery—the ansa subclavia—which is intimately related to the cervical pleura. The phrenic nerve is also related to the cervical pleura at the thoracic inlet. Adhesion between these structures would cause injury to one set of nerve fibers on traction on the other set.

53 Chittaranjan Avenue (12).

ACKNOWLEDGEMENT

I am indebted to Dr. K. L. Sen, professor of ophthalmology, Medical College, Calcutta, for permission to publish this case and to Mr. A. N. Das Gupta for the drawing.

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CICATRICIAL ECTROPION*

REPORT OF THE PLASTIC REPAIR OF A BILATERAL CASE

CHARLES V. BARRETT, M.D.
Evanston, Illinois

The treatment of mild cicatricial ectropion is familiar to all ophthalmologists. This condition may follow severe burns, lacerations, ulcers, gangrene, and other conditions.

This case is the most advanced of its type in my experience and nothing similar to it has been found in the literature. As such it is worthy of report.

* Presented before the Chicago Ophthalmological Society, December, 1953.

Case report

Mrs. G. R., a 48-year-old white woman, was first admitted to Evanston Hospital on January 26, 1953, with the chief complaint of recurrent, progressively enlarging lesions of eight years' duration involving the entire face, ears, and neck. At the time of onset she first noticed a discolored, crusted, itchy lesion on the forehead. This lesion persisted and progressed with healing and extension during the succeeding eight years, with much scarring over the entire face. Gradually each lower lid began to be pulled down and away from its normal position until finally the only traces of the lower lids were a few scattered lashes on the malar eminences. Above this, and over a broad expanse, the red glistening surfaces of the conjunctival membranes were seen. At no time was there any pain; only moderate itching.

Physical examination was essentially normal.

The globes were unremarkable except for some coalescent opacities over the inferior one third of each cornea. Above this the corneas were clear and glistening. Visual acuity of the right eye was 20/25; left eye, 20/25−2, without correction.

On the face and neck there was mutilating scarring with ulcerations and crusts; and soft nodules were seen within the scarred area and at the borders of the advancing lesions. The patient also had a deep ulceration about three inches in diameter with a scalloped border just below the right knee.

Fig. 1 (Barrett). Before operation.

Fig. 2 (Barrett). After operation.

This was typical, clinically, of a syphilitic gumma.

Serologic tests for lues were positive with a quantitative Kahn of 512 units. However, the symmetry of the facial lesions within the scar and the "apple-jelly" like nodules in the lesion suggested a diagnosis of lupus vulgaris. Thus, in spite of the knowledge of a positive Kahn, a biopsy of the facial lesion was taken and reported as lupus vulgaris because of the marked tuberculoid structure of the histologic section. The absence of typical tubercules and also the absence of necrosis in the tuberculoid units ruled out lupus vulgaris as a positive diagnosis. A syphiloderm may have a tuberculoid pattern such as was seen in the sections on this case, but typical "coat-sleeve" infiltration and thickening of endothelial cells were nowhere to be found. Thus, a definite etiologic diagnosis could not be made. On the basis of response to penicillin therapy and the markedly high quantitative Kahn, lues appears most likely to be the causative factor.

Tuberculin tests and spinal-fluid examination were negative.

Treatment

The patient was given 12,000,000 units of penicillin within 12 days, and all lesions healed within a month. She was also given an additional 1,000,000 units of penicillin daily for five days prior to admission for surgery.

On July 1, 1953, under general anesthesia,

surgery was performed. A circumlinear incision was made four mm. below the lower border of the right lower lid. This area was thoroughly undermined and as much scar tissue as possible was tediously excised, thus freeing the lower lid and allowing it to come up easily into normal position.

When the recipient site was prepared, a full-thickness skin graft was cut free-hand from the inner aspect of the right upper arm in accordance with a pattern previously determined. All possible fat was removed from the graft, which was then laid in position and secured by several interrupted 6-0 black-silk sutures. Intermarginal lid sutures were put in medially and temporally, using 4-0 black silk. A heavy pressure dressing was then applied over the eye, and the patient was returned to her room.

Postoperative care consisted of early moderate ambulation and intramuscular Dicrysticin for 10 days. The dressing was removed 14 days postoperatively, and a complete "take" of the graft was noted. A few sutures a day were then removed until all were out at the time of discharge.

It was my plan to leave in the intermarginal adhesions for six months but, in four weeks, the lids began to separate and, in spite of three more attempts, the lids could not be made to hold together long enough to do any good.

Healing thereafter was uneventful and, with the exception of a moderate cicatricial ectropion on the temporal aspect of the right upper lid, and a thickness and redness of the right lower lid, the result was quite satisfactory.

On February 9, 1954, the same procedure was performed on the left eye, using skin from the left arm as already described. The same postoperative course was followed except that the pressure dressing was changed after only one week. This time only the medial intermarginal sutures tore out prematurely, the lateral tarsorrhaphy remaining intact for six months, when the lids were surgically separated.

SUMMARY

I have presented the case of a patient whose cicatrizing facial lesion was neglected until it had reached an almost incapacitating stage. Absolute diagnosis could not be made but, on the basis of clinical experience, syphiloderm seems the most likely. The ease with which the grafts healed in so doubtful a site should encourage all who are faced with the problem of plastic surgery about the eye, and illustrate to the ophthalmic surgeon that he is capable of this work.

636 Church Street.

LEIOMYOIDOMA OF THE IRIS

REPORT OF A CASE

OGDEN D. PINKERTON, M.D.
Honolulu, Hawaii

Tumors involving the dilator myoid of the iris and those of the sphincter are relatively rare. Erdbrink and Harbert,[1] in reporting a leiomyoma of the iris, have reviewed the literature, and reference to their excellent article is recommended.

REPORT OF A CASE

A white woman, D. E., aged 31 years, a nurse, was examined on April, 1954, with a history of her left eye being recently bloodshot, and a constant sensation of pressure in the eye.

External examination was negative except for slight circumcorneal injection of the left eye and an easily visible fleshy mass in the iris periphery, at the 7-o'clock position, approximately 2.5 mm. in size. Her vision was 20/20 in each eye with a minus glass. The pupils were equal but the left had a suggestion of flattening in the same meridian as the tumor mass. Reactions to light and accommodation were normal.

Ocular tension (Schiøtz-electronic) was normal. Slitlamp examination revealed that the fleshy mass had an umbrellalike ar-

rangement of blood vessels which were mature in appearance.

The mass indented the iris posteriorly and appeared to extend into or possibly arise from the angle. This was confirmed gonioscopically. The trabecular structure of the angle was visible on either side of the mass only. There was a slight suggestion of scattered pigmentation in the growth.

Under mydriasis the iris dilated equally throughout its circumference. The anterior chamber and anterior lens surface were free of cells or pigment debris. Fundus examination was normal.

The history revealed that the patient had noted this growth for at least two or three years, and that she had been questioned about it by another oculist at that time. She vaguely felt that it had become more noticeable over the past several months. Two years previously she had several pigmented moles removed from her back and left axilla. She stated that her doctor had examined these sections and they were benign. Her history was otherwise noncontributory. She had recently had a complete physical examination which had included the routine laboratory studies—chest, X-ray, breast, and pelvic examination.

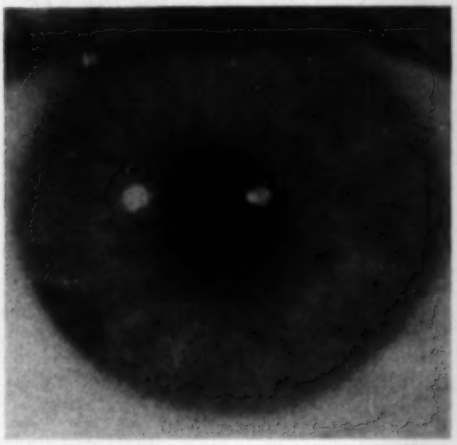

Fig. 1 (Pinkerton). Leiomyoidoma of the iris. Preoperative. Tumor in iris periphery at the 7:30-o'clock position.

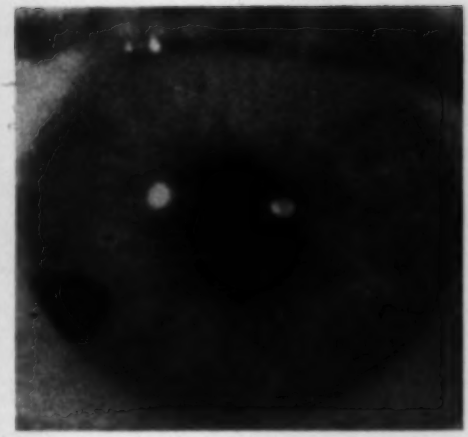

Fig. 2 (Pinkerton). Leiomyoidoma of the iris. Postoperative.

Between April, 1954, and August, 1954, she was examined by several consultants among whom were Dr. Merrill Reeh of Portland, Oregon, Dr. Samuel Meyer of Chicago, and Dr. Brittain Payne of New York City. All felt that careful and continued observation was necessary and that, if signs of growth should occur, excisional biopsy should be done.

In November, 1954, it appeared that the iris flattening—or deformity—had increased (fig. 1) and that the mass had increased in size. On November 4, 1954, under local anesthesia, a scratch incision was made 1.5 mm. behind the limbus at the 7-o'clock position. The tumor mass presented itself through the wound after massaging the adjacent sclera gently. It was grasped firmly by toothed iris forceps, pulled forward, and excised, apparently in its entirety, with two snips of the iris scissors. The postoperative appearance is shown in Figure 2.

Postoperative gonioscopy showed a clean and complete root iridectomy with the lens margin and ciliary processes clearly visible. No tumor mass was evident.

Biopsy studies by Dr. Merrill Reeh of Portland, Oregon, and Dr. Harold Civin of Queen's Hospital, Honolulu, were reported as follows:

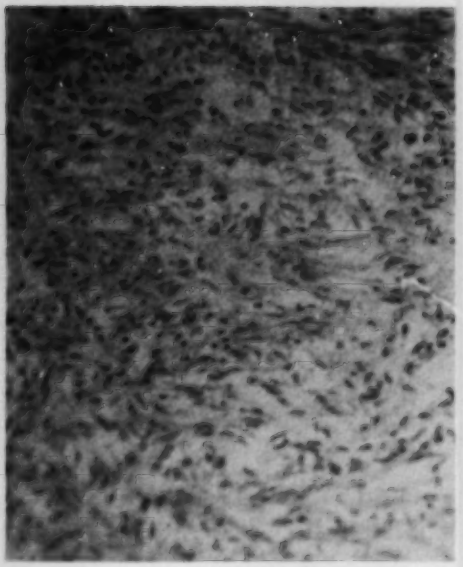

Fig. 3 (Pinkerton). Leiomyoidoma of the iris, showing spindle cells with oval nuclei. Some cells show a terminal filament or process.

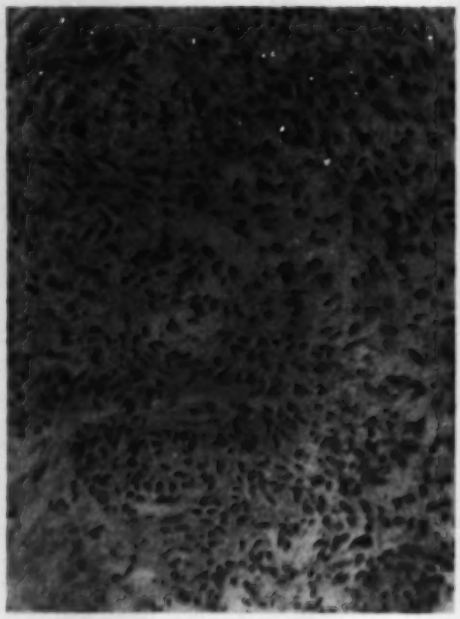

Fig. 4 (Pinkerton). Leiomyoidoma of the iris, showing palisades or fencelike lamina of spindle cells surrounding groups of cells.

"Histologic study of this specimen shows it to be composed almost completely of spindle-shaped cells with oval nuclei, some varying in thickness, with granular chromatin. There are some cells which do not have the characteristic oval nucleus. Many of the spindle-shaped cells demonstrate a fibrous process (fig. 3). They are either visible on one or both ends of the cells. Throughout the tumor there are groups of cells which appear to be surrounded by a fencelike lamina of spindle-shaped cells (fig. 4). There is a small amount of pigment scattered throughout the tumor."

Discussion

This tumor apparently arises from the myoepithelial cells of the dilator muscle of the iris, taking origin from the periphery of this structure. The position of the tumor, its appearance, and its tendency to extend upward from the periphery of the iris is in keeping with this type of tumor.

Dr. Reech presented this as a leiomyoidoma representing a tumor of *dilator* origin. This specimen section was studied by Dr. Parker Heath of Boston, who concurred in the diagnosis. Parker Heath[2] has classified and discussed neuroectodermal tumors of the iris. His classification is:

Sphincter muscle type	Dilator myoid
Benign	*Benign*
A. Leiomyoma	A. Benign epithelial proliferations; cysts
	B. Leiomyoidoma
Malignant	*Malignant*
A. Malignant melanoma	A. Malignant leiomyoidoma
B. Leiomyoblastoma	B. Leiomyoidblastoma Embryonal leiomyoma which contains some myoid elements (or medullary epithelioma with teratoid and retinal elements).

Reese[3] classifies and describes these neuroectodermal tumors and lays down certain criteria for diagnosis, with extensive

consideration of differential clinical and pathologic diagnosis.

A simple classification based upon tissue of origin is possible when differentiation has gone far enough. Subdivisions are then a matter of interpretation of atypical forms. The more primitive the tumors the more they resemble each other.

In general, tumors arising from the sphincter are more benign than those arising from the dilator. The latter are, in general, somewhat more rapid in growth.

230 Young Hotel Building (13).

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A MODIFIED REESE KNIFE*

FOR PTOSIS AND SIMILAR SURGERY

CONRAD BERENS, M.D.
New York

The correction of ptosis by means of attaching the skin of the eyelid or strips of orbicularis to the frontalis muscle have been employed by several authors. Reese,[1] in 1923, devised a double-edged knife with an eye which was used to tunnel a path beginning just above the crest of the eyebrow down to the prepared tissue flap above the eyelid margin. The disadvantage of the Reese knife was the fact that the blade was flat and when a side-to-side motion to form an adequate tunnel was required, there was no bevel to keep the knife in the same plane in the tissue. Furthermore, the tip was not beveled on the anterior surface to assist in keeping the knife from perforating the skin as it was thrust forward.

Because of these disadvantages, the largest size of the special skin-grafting knife,[2] which is hollow ground on the lower surface and beveled on the anterior surface, was used to make the tunnels in the upper eyelid, and then a probe with an eye was passed to which the sutures in the orbicularis were attached.

To facilitate the operation, a new slightly narrower and more tapered knife† length of blade, 36 mm., width of blade, 4.5 mm.) was constructed with a 1.5-mm. suture hole placed near the tip (fig. 1).

Fig. 1 (Berens). A modified hollow-ground beveled knife with a 1.5-mm. suture hole, useful in surgery of ptosis and for the excision of grafts of skin and mucous membrane.

The knife may also be used in any surgery requiring a tunnel, as well as for removing skin and mucous membrane, since the hole for the reception of the suture does not interfere with the passage of the knife.

708 Park Avenue (21).

* From the Department of Research, New York Association for the Blind, and the Department of Ophthalmology, New York University Post-Graduate School of Medicine. Aided by a grant from The Ophthalmological Foundation, Inc.

† Made by the Storz Instrument Company, Saint Louis, Missouri.

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DR. FREDERICK H. THEODORE, *President*

ROUND TABLE CONFERENCE: CATARACT

Discussors: DR. JOHN G. BELLOWS, DR. RAY K. DAILY, DR. JOHN M. MCLEAN, AND DR. HARVEY E. THORPE

Moderator: DR. FREDERICK H. THEODORE

1. *What is the underlying basis for the loss of vision in the three types of senile cataract? Is the loss of vision always in proportion to the degree of opacification? What nonsurgical treatment might improve such diminished vision?*

DR. BELLOWS said that the underlying mechanisms for the loss of vision in cortical, nuclear, and posterior saucer-shaped or cupuliform cataracts differ. The cupuliform cataract, beginning with vesicle and crystalline formation at the posterior pole just anterior to the capsule, will cause a marked reduction in vision early, and at a time when the remainder of the lens may still be very clear. In cortical cataract, on the other hand, the lens may have large opaque areas and yet if the axial area is clear, there may be surprisingly good vision. Not until the central region becomes opaque is there a marked reduction in vision. Finally, in nuclear cataract, the increased brownish coloration causes blue blindness while the progressive sclerosis increases the index of refraction resulting in lenticular myopia.

There is no satisfactory medical treatment for cataract. However, while waiting for surgery, the patient may be made more comfortable by correcting the increasing myopia and cylindrical changes. The patient with cortical cataract reads better with a light in front of him, while the patient with a nuclear cataract or a cupuliform cataract will benefit by having the light coming from the side.

2. *What is your postoperative regimen in cataract surgery?*

DR. DAILY said that the operated eye is patched after the instillation of pilocarpine and eserine. The unoperated eye is left open. The patch is replaced by a perforated metal disk on the fifth postoperative day. Dark glasses are substituted for the metal disk on the eighth day, and the patient instructed to wear a ring mask at night for several weeks. The eye is dressed on the second postoperative day, when the anterior chamber is usually found restored, and the pupil very narrow; a mild mydriatic is instilled. The dressings are then changed every day or every other day, and atropine used as necessary to keep the pupil moderately dilated.

The patient is placed in a chair 24 hours after the operation, and remains in the hospital for a week. He is restricted for a few days to a soft, salt-free, or salt-poor diet, depending on his cardiovascular condition, and the diet is supplemented by mixed vitamins by mouth. If the unoperated eye is useful, glasses are not fitted for four to six weeks. If the patient is nauseated, he is given Dramamine, although with a tight closure of the wound, nausea presents no great hazard. Because she believes that an anxiety-free attitude of the patient is an important factor in smooth postoperative recovery, she keeps her patients well sedated during the hospitalization period.

DR. MCLEAN said that he agreed essentially with Dr. Daily except for one thing. He does not like to give the patient more sedation than he is normally in the habit of taking unless there is some special indication for it. He also believes that there is no need to bandage the unoperated eye.

3. *Discuss the advisability of intracapsular versus extracapsular extraction in the presence of high myopia, old uveitis, posterior synechias, and atopic cataract.*

Dr. McLean said that this question is based on the old and, he believes, incorrect notion that the conditions which are listed in the question are indications for extracapsular extraction. He believes that they are indications for intracapsular extraction. The argument has been that in high myopia we usually have fluid vitreous and that that is a contraindication to intracapsular extraction.

Old uveitis is often accompanied by the presence of posterior synechias which are no contraindication to intracapsular extraction. Intracapsular extraction can be done frequently and, in fact, it is unusually easy in these cases, as these cases of old uveitis are prone to have weak zonules.

Posterior synechias are listed as a separate problem, but what he said before holds for them and atopic cataract, both of which are indications for intracapsular extraction. In these cases, we should be particularly anxious not to leave any lens protein. Some of these atopic cataracts are not easy to grasp with forceps.

In summary, Dr. McLean believes that in all these cases mentioned in the question, intracapsular extraction is distinctly in order and the old theory that extracapsular extraction should be done is both wrong and obsolete.

Dr. Theodore questioned: If you had a young patient with an old uveitis who had to have a cataract extraction, would you do it by the intracapsular method?

Dr. McLean stated that he would, as he thinks that the presence of old uveitis will tip the balance in favor of intracapsular extraction.

Dr. Daily and Dr. Thorpe agreed completely with Dr. McLean on the choice of an intracapsular extraction.

4. *What measures can be taken to reduce the incidence of vitreous loss in cataract surgery?*

Dr. Thorpe said that loss of vitreous is one of the most serious complications which may occur during cataract surgery. The loss of fluid vitreous is less serious than escape of the normal vitreous humor. Measures can be taken to reduce the incidence of vitreous loss during cataract surgery. These deal primarily with planning surgery so as to avoid any possible pressure on the globe either (1) by the patient's lids (due to poor akinesia or narrow palpebral fissure), (2) by the patient's extraocular muscles (both the recti and obliques), (3) by the lid speculum or retractors, or (4) by the surgeon's instruments or other breaks in technique.

The likelihood of the occurrence of the aforementioned condition can be reduced (1) by allaying the patient's possible apprehension and having him in proper mental state, (2) by adequate anesthesia, (3) akinesia, and (4) by the use of an adequate number of corneoscleral or scleral (hard tissue) sutures. This means a minimum of three such sutures. (His routine procedure is the use of five preplaced scleral sutures.)

The patient's co-operation is obtained, in a greater degree, if the surgeon is unhurried and relaxed. A light (small red or green bulb) for the patient to fix his gaze during surgery is helpful. This holds the patient's attention and avoids aberrant movements of the globe during the operation. Injection of the recti muscles with procaine or Xylocaine and the placing of local anesthetic in Tenon's space prevent spasm pressure by these muscles on the walls of the globe. Adequate akinesia, either by the method of O'Brien, van Lint, or Atkinson, avoids muscle pressure by the lids on the globe. A canthotomy lessens the pressure of lids on the eyeball in case of a narrow fissure. The use of hyaluronidase with the retrobulbar injection results in a softer eyeball. Firm pressure maintained on the orbital contents for five minutes after the retrobulbar injection also results in a softer eyeball and less likelihood of vitreous prolapse. The use of curare is helpful at times.

On the other hand, the use of intravenous succinylcholine has been reported experimentally by Lincoff as causing an increase in intraocular pressure due to spasm of the extraocular muscles. He has had a similar impression when he operated a few patients under general anesthesia with succinylcholine used as a supposed muscle relaxant.

In summary, mental relaxation on the patient's part, lack of tension and hurry on the surgeon's part, adequate anesthesia and akinesia, proper selection of instruments, avoidance of pressure on the globe by adnexal tissues or instruments, and an adequate number of hard tissue preplaced sutures are the prerequisites for avoidance of vitreous loss.

Dr. Daily commented that taking the intraocular pressure after the pupil is dilated before opening the eyeball may alert the surgeon to the danger of vitreous loss caused by an increased intraocular pressure. Keeping the patient asleep during the operation eliminates loss of vitreous caused by reflex pressure of the extraocular muscles. She accomplishes this by an intravenous injection of three grains of nembutal just as the operation is started. This usually keeps the patient quietly asleep for the duration of the operation.

Dr. McLean added that Dr. Thorpe had summed it up very well. A quiet eye without tension, a quiet patient without tension, and a quiet surgeon without tension are the triad that help to prevent vitreous loss. Dr. McLean remarked that the fixation light for the patient during surgery was very interesting. He would prefer to have such an adequate retrobulbar injection that the patient could not change his direction.

5. *What are the causes and treatment of postoperative flat anterior chamber following cataract extraction?*

Dr. Bellows said that flattened anterior chamber following cataract extraction is caused by a leaking wound that persists for over six hours. This sets into motion a series of events recognized clinically by the flattening of the anterior chamber and elevation of the choroid. The difference in the pressure of the anterior chamber, which is equal to that of the atmosphere, and the pressure posterior to the iris, which is relatively high, causes the vitreous to shift forward, blocking the pupillary and iridectomy openings.

As aqueous continues to form, it presses the vitreous more firmly against the iris and its openings. Later, back pressure develops against the ciliary body and processes resulting in an interruption in the formation of the aqueous. A similar back pressure against the choroidal venous sinuses leads to stagnation and edema with transudation into the suprachoroidal space resulting in the elevation of the choroid. This blocking explains why it is that by the time the surgeon recognizes the condition, usually the first postoperative day, a negative Seidel fluorescein test is obtained even though a wound defect may be present.

In most cases spontaneous restoration of the chamber occurs. This is brought about by the interruption of the formation of aqueous and the absorption of the accumulated aqueous through the posterior routes. The pressure upon the vitreous and its obstruction of the pupillary opening are thus relieved, permitting the aqueous to flow into the anterior chamber.

Dilatation of the pupil with atropine may be beneficial in helping to overcome the block, since a large opening is obstructed with more difficulty than a smaller one. If the chamber does not reform in three days, Diamox is administered. This helps by further reducing the aqueous formation and thus reducing the pressure posteriorly. If Diamox fails, an air bubble is introduced into the anterior chamber, and if this procedure is also ineffective, ophthalmologists up to now have performed posterior sclerotomy. Since this reduces the pressure in the vitreous chamber it is frequently effective.

Recently, he performed an incision through the anterior hyaloid of the vitreous, entering through the cornea in a manner

similar to a discission operation. This results in an immediate restoration of the anterior chamber as it fills with vitreous and aqueous. In three cases thus treated uneventful recovery took place.

Dr. Daily added that, since she has been suturing the keratotomy wound firmly, delayed reformation of the anterior chamber is an extremely rare occurrence, except after removal of the sutures; and then it is restored spontaneously in one or two days. Before she sutured the wounds so thoroughly, she used to see more of them, and she dealt with them by the injection of air into the anterior chamber. A number of times she had a rather puzzling experience; after preparing the eyeball for the air injection by anesthetizing the eyeball, akinesia, and retrobulbar injection, it was found that the anterior chamber had restored on the table, while waiting 10 minutes for the Novocain to take effect. This made her wonder if some metabolic process in the vitreous, or the vegetative ocular innervation, was a factor in cases in which no leakage could be demonstrated.

Dr. McLean said he agreed with Dr. Bellows completely that leakage through the wound is the cause of flat chamber and he also agreed with Dr. Daily that in the past, with relatively inadequate wound closure, flat chamber was well known and we used to wait several days before the anterior chamber was reformed. Now on the first dressing we expect a deep chamber. Sometimes a flat chamber will reform while you are making up your mind, sometimes while you use mydriatics. He believes that the most useful tool of all in flat chamber is air inflation into the anterior chamber. That is so much simpler to do without posterior sclerotomy. He believes it is worthwhile to do a simple air injection first.

Dr. Thorpe said that he too has been anxious to avoid leakage from the anterior chamber. For a number of years he has closed the cataract incision with sutures, placed so as to approximate the corneal wound margins evenly. It has been his routine practice to restore the anterior chamber at the time of the surgery by the instillation of an air bubble. In spite of that, he found that about four percent of patients having cataract extraction developed a flat chamber at some time during the convalescent period (sometimes as late as the 10th day after cataract extraction). These patients usually had an early choroid detachment. It was not possible to demonstrate or see an actual leak with the biomicroscope and fluorescein in most of these cases. Incisional leaks occurred only on rare occasions and in spite of multiple preplaced scleral sutures and a limbus-based conjunctival flap. That very deep sutures may cause a wound leak and that too superficially placed sutures may cause deep wound gaping has been pointed out by Dunnington and others.

To restore nonleaking flat anterior chambers, surgeons have practiced injection of air and drainage of suprachoroidal effusion. Dr. Thorpe has abandoned this method in favor of oral administration of Diamox in large dosage. It has proven to be helpful in the restoration of nonleaking flat anterior chambers associated with early choroidal detachment. Diamox apparently causes a reduction of aqueous secretion. The diuresis induced by Diamox is probably responsible for reduction of the choroidal detachment. Reduced aqueous production is not sufficient to prevent chamber restoration as the choroid flattens.

Most of our cases with flat postoperative nonleaking chamber with early choroidal detachment became re-established within a few hours or within several days after Diamox therapy was commenced. On the other hand, a few patients were refractory to Diamox. There are reports in the literature that Diamox failed as a diuretic in an occasional case of congestive heart failure, but that it was potentiated by mercurial diuretic administration. Similarly, Dr. Sachs of Boston reported to him Mercuhydrin injection helped in flat chamber restoration

in a case in which Diamox failed.

There are many remedies in medicine and none of these remedies has a 100 percent desired beneficial effect on patients. We should keep an open mind and try Diamox in flat nonleaking postoperative chambers. He believes it to be useless in cases with frank leak in the incisional wound.

6. What are the important factors influencing corneal wound healing after cataract extraction?

Dr. Daily said that the most important factors are a wound free of incarcerated tags, and perfect apposition of the wound edges. In 1947 she came across an article in the *British Journal of Ophthalmology* on the repair of ocular wounds, which had some novel ideas. The pathologist demonstrated with microscopic sections that wounds which are not firmly sutured are covered by epithelium on the external surface, but remain separated in the deeper layers. In the process of repair, one sees always on the posterior corneal surface the figure of a triangle, with the base toward the anterior chamber; the sides represent the edges of the corneal wound. This space is quickly filled up with newly formed tissue growing from the edges of the wound and getting more profuse toward the anterior chamber where the edges are more widely separated. This tissue may proliferate and form whole layers on the posterior corneal surface.

Defects of the endothelium or surface of the iris appear to stimulate these growing cells to a powerful process of overgrowth. The growing tissue may encapsulate the anterior chamber and penetrate deep into the globe. When the edges of the wound are correctly joined, the repair of the wound takes place by a growth of this tissue within normal limits, and an overgrowth does not occur.

The intriguing feature of this article was the claim that repair of corneal and scleral wounds does not take place by the process of repair of connective tissue formation, but by a proliferation of its own cellular elements that morphologically resemble tissue cultures. If these cellular elements penetrate into the anterior chamber, they encounter all the favorable conditions for tissue culture, such as a liquid medium, temperature, nutritive substances, added by fibrin from hemorrhage and its solution. The rise in tension or retinal detachment, which finally causes the destruction of vision, are processes secondary to the invasion of the eye by this exuberant tissue.

Dr. Daily was intrigued by this article enough to go over, in her own collection, the microscopic specimens of eyes enucleated following unsuccessful cataract extractions. The eyes were sectioned at the Army Medical Museum, and the diagnosis was mostly traumatic iridocyclitis. The most interesting specimen was that of the left eye of a 26-year-old man, enucleated three and one-half months following discission for a traumatic cataract. It shows a very interesting formation—a plaque of tissue on the posterior corneal surface, indistinguishable from corneal stroma.

Several slides were shown illustrating the tendency of this proliferative fibrous tissue to invade the interior of the eyeball. We therefore have to consider the probability that what we have been calling postoperative iridocyclitis is really a destructive proliferative process, caused by a gaping keratotomy wound. She uses five corneoscleral sutures to close the wound firmly and cover it with a limbus or fornix based conjunctival flap. With exact apposition of the wound edges, she is not able to tell much difference in the healing of wounds covered by limbus based or fornix based conjunctival flaps.

7. (a) *Should a patient with a cataract in one eye with a normal fellow eye be operated upon?* (b) *What is the present status of the Ridley and the Bietti lenses?*

Dr. McLean said (a) the answer is sometimes yes, and sometimes no, but more often yes than no. It depends on the individual situation. We must remember that a

patient with a cataract in one eye, if he is left alone for a long period of time may have secondary glaucoma, or lens induced uveitis. He is likely to develop heterotropia and it may be very difficult at a considerably later date to rehabilitate that eye, whereas, in most instances, the eye can be operated on earlier in the stage of the development of the cataract with a good result and the patient can usually be rehabilitated with contact lenses with a good chance of binocular vision. It was Dr. McLean's belief that in most cases the patient deserves the benefit of an opportunity to have two good eyes which is denied him if one insists on saying that if he has one good eye that the other one should not be operated.

(b) In the opinion of Dr. McLean, the Ridley lens and the Bietti type of lens, at the present time, should still be considered as experimental. This is entirely his opinion, but he does not believe at the moment that we are justified in using them on patients, unless the patient understands that they are not as yet proven procedures which have stood the test of time. If a patient wishes to volunteer for an experimental procedure and it is done as a controlled experiment, he believes it is justifiable.

Dr. McLean feels that the Bietti type of lens offers much more promise than the Ridley type of lens. It would appear to be safer. It does not necessarily have to be introduced at the time of operation. It could be introduced a long time after. It should be much easier to remove. At the present time, however, he felt it was safer for the patient to have the plastic foreign body on the outside of the eye than on the inside. The future probably will tell about intraocular lenses. Dr. McLean is not so sure it is going to be the Ridley type. He is quite certain in his own mind that it is not a procedure to be undertaken lightly at the present time.

Dr. Thorpe said he thinks Dr. McLean adequately answered the question of surgery of single cataract in a two-eyed patient by his first remark "sometimes yes and sometimes no." The patient must be evaluated from the standpoint of age, health, occupation, and state of cataract maturity. There should be little objection if the fellow eye shows a partial lens involvement and the affected eye has a mature cataract. One must explain to the patient that he will not have binocular vision by conventional means. As to the other question, plastic lens implantation is still in the experimental stage.

Dr. Daily added that she has done four cases with the Ridley lenses. Two were in young people and they did very well; two in senile cataracts, which were followed by a stubborn postoperative reaction, and poor functional result. She believes that these procedures are still in the research stage, and the last word about them has not been said.

8. *How would you treat iris prolapse:* (a) *on the day following cataract surgery,* (b) *a week after cataract extraction?*

Dr. Thorpe said that more iris prolapses occurred before the day of multiple preplaced scleral sutures. The treatment of iris prolapse one day after cataract surgery now consists of an attempt to replace the prolapse with placement of an additional corneoscleral or scleral suture. Miotics to reduce the prolapse have proved inadequate in his experience. When iris replacement is impossible, it is freed and excised. The wound is freshened and a corneoscleral suture inserted.

In prolapse occurring a week after surgery, there is no urgency if the prolapse is small and is covered by conjunctiva. One can wait to see whether the prolapse has any tendency to enlarge. The patient can sometimes go along with a small prolapse. If there is any tendency for the prolapse to enlarge, or if it is not covered by conjunctiva, he finds it necessary to excise the extruded iris knuckle. It must be freed from the wound margins. The wound edges must be freshened and sutured.

Dr. Daily said that she had never seen

miotics effective in reducing an iris prolapse.

DR. MCLEAN said that he has occasionally seen an iris prolapse pulled back in with a combination of miotics and adrenergic mydriatics in an attempt to tense the sphincter and dilator simultaneously. He added that often that attempt is disappointing.

9. *What are the causes and treatment of hyphema following cataract extraction?*

DR. BELLOWS said that, at the time of the operation, the conjunctival flap, iris, choroidal vessels, and the corneoscleral incision can be the source of hyphema; later, during the postoperative course, the source is usually rupture of the newly formed vessels that span the corneoscleral section.

In the prophylaxis of hyphema associated with cataract surgery, precision in corneoscleral suturing appears to be an important factor in reducing the incidence of bleeding. The value of other prophylactic measures is questionable. Vitamin C does not seem to be important because hyphema appears even in the presence of high vitamin-C blood levels. Vitamin K is of value only when intestinal and hepatic disease are present.

Hyphema arising at the time of operation is treated by coagulation of the bleeding points with a heated applicator. Any blood is removed from the anterior chamber by irrigation. If a subchoroidal expulsive hemorrhage is impending, a posterior sclerotomy is urgently indicated. The hyphema that appears four to seven days after operation is treated by elevation of the head, bedrest, and close observation. If the hemorrhage is slow in absorbing, pilocarpine is administered to increase the absorptive surface of the iris. In the rare case of late hemorrhage from the iris, atropine is instilled to immobilize the iris and ciliary body. If blood should fill the entire chamber, paracentesis is indicated.

10. *What are your indications for operation in congenital cataracts? At what ages do you operate and what procedures do you prefer?*

DR. DAILY said that in total bilateral cataract one eye may be done when the child is six months old, and the other later, guided by the result in the first eye. In unilateral cataract there is no indication for early operation, because the eye usually remains amblyopic anyway. In partial cataract, we wait until the child is about five years old, when its visual acuity can be determined. If it is below 20/50 surgery is indicated. The results have been better after discission, repeated if necessary. Usually, both eyes are operated on. In view of the tendency of these eyes to develop retinal detachment, there is probably good justification for operating on one eye only.

11. *Would you vary your technique in an extremely elderly, somewhat debilitated patient?*

DR. MCLEAN replied, only slightly. He would be particularly careful to put in a large number of corneal sutures, to get the patient into early ambulation, and to use an absolute minimum of sedation. Otherwise there would be no change.

12. *What is your choice of sutures in cataract surgery?* (a) *Type of suture material and needles?* (b) *Corneal, scleral, conjunctival?* (c) *Pre- or postplaced?*

DR. THORPE prefers No. 82-7 Grieshaber needles and braided black 6-0 silk over catgut or nylon suture material. The new polished American-made atraumatic needles have good sharp points and also work well. He uses five preplaced scleral sutures with a limbus-based conjunctival flap. The scleral incision with scleral sutures has, in his experience, reduced the incidence of keratitis striata during convalescence.

With this type of incision and suture it has become possible to operate patients having endothelial dystrophy or early Fuchs' dystrophy without rapid postoperative aggravation of the corneal condition. Patients can thus be safely permitted to leave the unoperated eye open and to leave the bed four or less hours after surgery.

As stated, his preference is for five preplaced scleral sutures and a limbus-based

conjunctival flap. The conjunctival flap is closed with a running suture.

DR. DAILY added that for the last year she has been using 6-0 catgut on Grieshaber needles, with very satisfactory results.

DR. MCLEAN said that he is always trying to improve sutures. He thinks that the best theoretical closure is obtained with the Verhoeff track suture. He usually uses the one that he thinks is the next best substitute. He does not agree with some that a single corneoscleral suture is adequate. He thinks two is a bare minimum. He wishes there was a sufficiently manageable strong gut that he could use in a preplaced suture.

13. *How are the different forms of complicated cataract differentiated from their counterparts in the senile cataract group and why is it important to do so?*

DR. BELLOWS said that different forms of complicated cataract are differentiated from their counterparts in the senile cataract group by a history of the case and by the fundus and slitlamp findings. Thus, the senile posterior saucer-shaped or cupuliform cataract and the posterior form of complicated cataract which appears in diseases of the posterior segment, as in retinitis pigmentosa, have a common site of origin, that is, the posterior pole directly anterior to the capsule.

The complicated cataract is mainly distinguished by the multicolored iridescences and the crumblike, white opacities which progress by rapid spurts, both axially and peripherally, along the suture system.

In high degrees of myopia and in commotio retinae, complicated cataract may appear in the form of a nuclear cataract. This type is differentiated from the senile nuclear cataract by history and fundus findings.

The complicated forms of anterior cortical changes which occur as a result of corneal or anterior uveal disease and in neurodermatitis must be differentiated from senile anterior cortical changes. Corneal scars, deposits on the lens surface and cornea, and synechias are found in the complicated cataracts caused by anterior segment diseases, and obvious skin changes are present in the dermatogenous type of complicated cataract.

Finally, complicated cataract occurring as a result of glaucoma must be differentiated from secondary glaucoma arising from intumescent cataract or from seepage caused by a break in the capsule.

Differential diagnosis is important because the prognosis in the complicated cataract differs considerably from that in the senile forms of cataract. Further, the prognosis varies in the different forms of complicated cataract. Extraction of the complicated cataract in eyes with retinitis pigmentosa, controlled glaucoma with good central vision, or in old cases of iridocyclitis, is frequently successful and permits the patients to resume a normal life. On the other hand, the result of the removal of a complicated cataract associated with diseases of the posterior segment, in which the central vision has been destroyed, will be disappointing.

14. *How would you manage a cataract in the presence of active uveitis?*

DR. DAILY said that following a granulomatous uveitis, it is well to wait two years before operating on a uveitic cataract. Phacogenic uveitis calls for immediate extraction of the lens.

DR. THORPE added that there is an exception to the generalization here. Cases of heterochromic cyclitis are treated for a few weeks before surgery by local cortisone instillation. They usually respond well to intracapsular surgery. There is no more than the usual amount of postoperative reaction as a rule.

DR. MCLEAN said that we could generalize by saying that the management of the cataract in the presence of active uveitis should be nonsurgical. There are three exceptions: incipient lens induced uveitis which is best treated by removal of the lens; heterochromic iridocyclitis; and lens induced glaucoma which may not respond to nonsurgical means and may require lens surgery.

15. *What are the indications for steroid*

therapy (cortisone, hydrocortisone, ACTH, and so forth) in cataract surgery?

Dr. McLean said that the indications for steroid therapy are very few and far between. He does not believe there is any justification for routine use of steroid therapy. It probably does lower the eyes' defense mechanism against infection and it probably lowers its resistance mechanism in other ways. There are indications for using steroid therapy in cataract surgery and they are: occasionally to suppress active or potentially active uveitis and also to give more safety in cases of long-standing, smoldering uveitis. Even though such an eye may have become quiet, he believes it is safer to do the lens extraction under steroid therapy as a prophylactic measure against recurrence. The other one situation: it is safe to do it if the patient requires steroid therapy for some nonocular disease process.

Dr. Thorpe said that in cases that have intraocular foreign bodies with traumatic cataract, he found steroid therapy extremely useful—particularly, ACTH intravenously. This reduces the endophthalmitis, and one can go ahead with the operative procedure. There has not been any noticeable interference in wound healing. In regular cataract extraction followed by unusual local reaction, we have used ACTH parenterally as early as the second day after surgery without untoward effect. The local use of corticoids when indicated is begun one week after surgery. It has been our practice to employ the preparation combined with a wide spectrum antibiotic.

16. *What is your procedure for cataract extraction following a successful filtering operation for glaucoma? Would you ever do an extraction as well as an iridencleisis at the same operation?*

Dr. Thorpe said that an intracapsular cataract extraction is routinely performed when one must follow a successful filtering operation for glaucoma. He prefers to avoid the area of previous surgery. He uses the method of corneal section immediately below the filtering area with preplaced suture as well as the inferolateral approach. He prefers the inferolateral scleral incision into the anterior chamber in the presence of a filtering scar above. A peripheral iridectomy is performed temporally when necessary to free posterior synechias. In several bilateral cases of this type he has performed the corneal section below the filtration bleb in one eye and the inferotemporal approach in the fellow eye. The latter proved to be more satisfactory. On occasion he has performed an iridencleisis at the time of cataract extraction. In recent years, he has avoided this procedure and has preferred to do a cyclodialysis at a later date if the tension rises.

Dr. McLean said that he has tried the section in clear cornea above in front of the filtering bleb, the temporal, and the inferior approaches. Unlike Dr. Thorpe, he finds that it is a little bit better to use the clear corneal section. His experience with iridencleisis is like Dr. Thorpe's.

17. *How does the age incidence of senile cortical cataract differ from nuclear cataract? What are the clinical implications?*

Dr. Bellows said that the age incidence of senile cortical cataract differs sharply from that of nuclear cataract. Incipient senile cortical cataract is usually observed about the middle of the sixth decade, perhaps even a few years later in the male. The cortical opacities if progressive will cause the patient to seek the aid of the ophthalmologist within 10 years. In short, the peak of incidence of cortical cataract occurs between the 65th and 70th year of life. If the individual has not developed a cortical cataract by the end of the seventh decade, it is unlikely that he will ever develop one. In contrast to this, the incidence of nuclear cataracts increases with age and these become the predominant type of cataract in the extreme elderly patient.

The clinical importance of these observations is that in the younger group with the soft, yielding, cortical cataract, a round pupil extraction is more easily accomplished

and a smaller corneal section is required than in the older group with the firmer and larger nuclear cataract.

18. *What criteria do you use in deciding whether to perform a round-pupil extraction or extraction with complete iridectomy?*

Dr. Daily said that a complete iridectomy is indicated if the operation is complicated by vitreous loss. Otherwise a round pupil is of advantage not only from an optical standpoint, but it also protects from complications. The intact iris holds the hyaloid back away from the cornea and the wound, and in extracapsular extraction it keeps the edges of the lens capsule from being incarcerated into the wound. In uveitis cataract, I think that it is well to do a sphincterotomy at the 6-o'clock position in addition to an iridotomy above.

Dr. McLean said that there are few indications for full iridectomy. They include rigid pupils, extensive posterior synechias, and central corneal opacities.

Dr. Thorpe stated that 90 percent of his patients under 60 years of age are operated with round pupil and peripheral iridectomy.

19. *Would you extract a cataract in the face of a suspected retinal detachment in the same eye, and, if so, how would you do it?*

Dr. McLean said he had done it in the past, and he is still doing it. If you hope to operate successfully on detached retina, it is usually necessary to get the lens opacity out of the way. When lens opacity is relatively slight, it may interfere with adequate visualization of the retinal details, and the denser the lens opacity, the more true that statement becomes.

20. *What measures can be taken to minimize the possibility of epithelial downgrowth into the anterior chamber?*

Dr. Thorpe said that the chief measures for preventing epithelial downgrowth into the anterior chamber are: (1) adequate and clean closure of the eyeball incision, (2) covering the incision with conjunctiva. (3) preventing the turning-in of any conjunctival tags, (4) making certain that sutures do not traverse more than half the thickness of the cornea or the sclera. Sutures can also be buried beneath the limbus-based conjunctival flap. In that fashion, no epithelium can grow down along the suture track.

Jesse M. Levitt,
Recording Secretary.

Ophthalmic Miniature

Now the canal of Schlemm communicates mainly with veins within the sclera and episclera which are necessarily under lower pressure than the uveal and retinal veins, so that the pressure within the canal must be lower than the intraocular pressure. The peculiar structure of the ligamentum, I take it, serves in a most perfect manner to resist the intraocular pressure, thus keeping the lumen of Schlemm's canal widely open and permitting the aqueous humor to drain into it.

F. H. Verhoeff,
The pathogenesis of glaucoma,
Arch. Ophth., **54**:27, 1925.

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EPIDEMIC KERATO-CONJUNCTIVITIS

The suggestion that epidemic keratoconjunctivitis was a virus infection was first made by Wright,[1,2] who, in his study of an outbreak in Madras, was able to demonstrate what he called inclusions in corneal epithelial scrapings; and this material could be used to transfer the disease from man to man. Attempts to isolate any infective agent in culture or by transmission experiments in animals were unsuccessful.

In 1941 an epidemic of great economic importance broke out in the shipyard workers of the west coast of the United States and gradually spread through most of the country. Working with the scrapings from one of

* Reprinted with permission from *The Lancet,* January 28, 1956.

these patients, Sanders[3] claimed to have isolated a virus by intracerebral passage in mice. This virus could produce the disease in a human volunteer and was neutralized by convalescent serum from patients with the disease. After two or three years the major epidemic wave died down and only sporadic outbreaks were reported; from these the Sanders virus was rarely isolated. It is interesting to note that in many of these sporadic outbreaks the common connection between those infected was a visit to an ophthalmologist for some other condition, and on occasion it seemed that an instrument may have been the means of transfer—for example, all patients infected may have had tonography performed.

During 1951 and 1952 work on the Sanders virus[4, 5] made it clear that it was a variant of the St. Louis encephalitis virus; and it was also shown that sera from the local epidemics then occurring no longer neutralized the virus.[6] In contrast to the experiences in the United States, claims to have isolated a virus similar to the Sanders virus have been made in Turkey[7] and in Japan.[8] In both cases the virus caused an encephalitis in mice, and Sezer was able to adapt his virus to produce lesions on the chorioallantoic membrane of the chick embryo.

Despite the isolation of virus by Sanders and others, there was considerable doubt about the importance of these agents in the disease. Recently the condition has been reinvestigated by two groups of workers—Fowle[9] and her colleagues in Canada and Jawetz[10] and his associates in California. Both these groups have succeeded in isolating viruses of the APC group from their patients. In Canada the virus appeared to be an atypical form of the type-3 strains which have been shown by Ryan and others[11] to be the cause of pharyngoconjunctival fever; and Jawetz's strain seems to be a new type which has been called the Trim or type-8 virus. Serologic investigation of the type-8 virus[12] showed that 25 cases of epidemic keratoconjunctivitis occurring between 1951 and 1955 all showed a convalescent serum titer of 1 in 10 or greater, and only two of 29 sera from other eye conditions investigated by Jawetz showed such titers.

It seems that there is a definite association between epidemic keratoconjunctivitis and these APC viruses; their known resistance to antiseptics might well explain how transmission occurs by instruments used in ophthalmic practice. There is, however, a certain amount of evidence that other agents may also produce the disease; for not only are the viruses isolated by the early workers very different from the APC group, but there is also a difference in the clinical picture. In the more widespread epidemics seen by Wright and by Sanders there were definite constitutional symptoms, which the later workers do not mention. Thus, it is possible that the condition may have more than one cause, as is borne out by the occasional isolation of herpes-simplex virus from these cases.

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CORRESPONDENCE

Moore's lightning streaks

Editor,
American Journal of Ophthalmology:

In the May, 1956, issue of The Journal, Dr. F. H. Verhoeff refutes the arguments of four other authors who contest his opinion

that Moore's lightning streaks are innocuous.

For some time I had noticed faint vitreous opacities in both my eyes, which were sometimes apparent but usually ignored. On June 4, 1952, however, I was startled by the appearance of a compact opacity in my right eye, a bit temporal and above the point of fixation. Although my central vision was not impaired, the opacity was annoying.

The following day I suffered a new and unexpected annoyance. While testing the vision of patients and turning my head from the board to the patient and back, a flash of light appeared in the temporal upper periphery of my right eye with each movement of my head. This unpleasant phenomenon did not recur during the following days but the opacity persisted.

The coincidence of the sudden appearance of a fresh vitreous opacity and the flashes of light on movement of my head made me wonder if there was a connection between them. Since, at that time, I knew nothing of the work of R. F. Moore nor of F. H. Verhoeff on this subject, I had no proof of the connection.

Later, during the evening of November 10, 1953, while writing on the typewriter, I was suddenly harassed by a dense opacity floating in front of my left eye. My curiosity as to whether or not flashes of light would follow was promptly satisfied. On the next day, light flashes appeared in the temporal upper periphery of the left eye. However, they persisted only that one day in contrast to the opacity which did not disappear.

Former observers reported much longer periods for the persistence of the streaks—six weeks to nine years. In addition, they were later able to elicit the flashes at will. In my case, this phenomenon was limited to a single day and could not be produced at will. These were the only differences. I do not doubt that I suffered genuine Moore's lightning streaks in each eye. Up to now no serious consequences have ensued; especially was I spared from dreaded detachment of the retina. I am in the happy position of being able to confirm Dr. Verhoeff's conviction of the innocuousness of Moore's lightning streaks.

(Signed) E. Raubitschek,
Baghdad, Iraq.

BOOK REVIEWS

REGENERATION IN THE CENTRAL NERVOUS SYSTEM. (By thirty-three contributors.) Edited by W. F. Windle, Ph.D., Sc.D., Springfield, Illinois, Charles C Thomas, 1955. 298 pages, 68 illustrations, bibliography, index. Price: $9.50.

This is a fascinating book primarily of interest to the neurologist and neurosurgeon of course. However, here and there as you read, there are the reported results of meticulous experiments that should intrigue the ophthalmologic reader. For instance, in Chapter 1, "Regeneration of the central nervous system in fishes," by T. Koppany, we learn that Rasquin reported that five of 12 fishes, with their optic nerves cut, regained vision as judged by stern criteria. "She also made the very interesting observation," says the author of this chapter, "that surgical removal of the lens or lenticular degeneration made functional regeneration of the optic nerve impossible." We are left with no explanation or theory for this astounding finding.

The book is the product of a conference held in 1954 under the auspices of the National Institute of Neurological Diseases and Blindness under the chairmanship of Dr. Louis B. Flexner. The contributors are some of the world's leading investigators in this field. They discussed their studies of central nervous system regeneration in lower animals and man, and described their difficult and often tedious experiments in exciting fashion. In his foreword, Pearce Bailey, the able director of the institute, says, ". . . regeneration can occur in the central nervous system, which eliminates the feeling that some negative law of nature forbids regen-

eration. Gone, I believe, is the firm, obligatory and almost economical slogan, which has prevailed neurological educators for almost half a century, that central nervous regeneration does not occur."

The studies do encourage much optimism for the future, but ever so much more needs to be done. It is interesting to note the important role that pyrogenic substances and the steroids play in the success of some of the experiments. It is also interesting to note that our ophthalmic instruments are widely used by the investigators, whose surgical techniques perhaps could excell those of the most expert ophthalmic surgeon.

Derrick Vail.

PROCEEDINGS OF THE ALL-INDIA OPHTHALMOLOGICAL SOCIETY. Volume XIV, Session 1954. Printed at the Diocesan Press, 1956.

The major portion of this reprint of the *Proceedings* of the All-India Ophthalmological Society's 1954 meeting is concerned with a rather extensive symposium on retinal detachment. Various papers deal with the etiology, pathogenesis, diagnosis, medical problems, surgical treatment (including scleral resection), and general observations on the diagnosis and treatment of this condition. One is impressed with the completeness and general adequacy with which the subject is handled.

Additional papers deal with a variety of subjects. A paper on the "Modern treatment of trachoma by Chloromycetin and terramycin" by Dr. Satya Pal Gupta is particularly interesting. He recommends the complement fixation test as the only reliable laboratory diagnostic test for trachoma and describes in detail the method of making this test. He found both of the drugs under study to be effective in the first and second stages of the disease and recommends the ointment form applied four to six times a day.

This publication does much to demonstrate the caliber of ophthalmologic practice in India and the society is to be commended for printing these *Proceedings*.

William A. Mann.

TECHNICAL RESEARCH AND BLINDNESS. By C. G. Ritter. New York, American Foundation for the Blind, 1956. 40 pages. Price: 50 cents.

A large proportion of blind persons retain varying degrees of residual vision. Perhaps a third within the American definition of blindness can be enabled to read newsprint with optical aids, provided the motivation is present. Their use is no longer hampered by the fear of eye damage from eye use. Individual requirements influence the choice of aid, but most of the partially sighted can be helped by standard lenses of high strength. For the deaf-blind, the duties of the eyes and ears are shifted to the sense of touch. Dr. Gerritt van der Mey of Holland, though deaf-blind, earned a Ph.D. in mathematics—a remarkable illustration of the potentialities of such transference.

James E. Lebensohn.

SYMPOSIUM ON THE ROLE OF SOME OF THE NEWER VITAMINS IN HUMAN METABOLISM. New York, National Vitamin Foundation, 1956. Paper bound, 137 pages. Price: $2.50.

This brochure contains 11 papers given by distinguished scientists at a symposium held at Vanderbilt University School of Medicine in October, 1955. The discussion was limited to reports on pyridoxine, tocopherol, and pantothenic acid.

The dietary requirement for man ranges from a clear dependence for ascorbic acid to much less defined needs. Where man is known to synthesize vitamins, the synthesis is from specific precursors—vitamin A from carotene, niacin from tryptophan.

The unravelling of the vitamin-B complex started with the isolation of riboflavin in 1933. Nicotinic acid amide was identified

with the antipellagra factor in 1936. Pyridoxine, which was isolated in 1938, acts not only as a vitamin but also as a proenzyme and, as such, is important in the utilization of tryptophan and cysteine. The precise role of tocopherol and pantothenic acid is still in dispute. Though animal experimentation suggested the use of tocopherol in progressive muscular dystrophy and sterility, this therapy proved valueless. It may play a part in maintaining the integrity of the erythrocyte. Evidence indicates that pantothenic acid is a metabolic essential but that a deficiency thereof implicates the adrenal cortex seems dubious.

James E. Lebensohn.

MIKROPSIE UND MAKROPSIE: A CLINICAL-PSYCHOPATHOLOGIC STUDY. (Bibliotheca Psychiatrica et Neurologica, Fasc. 98.) By Christian Müller. Basel, S. Karger A. G., 1956. 60 pages, bibliography. Price: SFr.: 8.50.

Dysmegalopsia is a relatively rare phenomenon. The ophthalmologist is primarily concerned with the peripheral form, that is, involvement of the retina or choroid. Micropsia is due to an edema causing separation of the cones. Macropsia, on the contrary, is the result of scar formation in the macular area with retraction of the perceptual elements of the neuroepithelium.

Disturbances of the accommodative mechanism are frequently mentioned in the literature as the cause of dysmegalopsia, although not substantiated by statistics or even individual case reports.

Dysmegalopsia may be a symptom of a localized brain lesion—either a tumor or a vascular disturbance. It is of little significance as a localizing sign, although the temporal lobe seems to be involved more frequently than other regions. Occasionally, dysmegalopsia is part of the aura in idiopathic or Jacksonian epilepsy.

The best known form of dysmegalopsia is the exogenous, toxic type; alcohol, cocaine, and, in particular, mescal are well-known agents. These illusions are quite characteristic because there seems to be a continuous change in the size of the objects during the attack. Even though the patients experience these illusions almost as in a dream, they never lose a critical insight.

The dysmegalopsia of the endogenous psychoses, especially schizophrenia, is quite different: it is either associated with hallucinations, or it is itself of a hallucinatory character.

Occasionally, dysmegalopsia is encountered in neuroses. There is some doubt whether it ever occurs in psychically healthy persons, unless caused by abnormal stress, that is, hypoxemia or fatigue.

The analytic evaluation, though a major part of the brochure, is of only limited interest to the ophthalmologist. It is doubtful that dysmegalopsia could ever pose a diagnostic problem. Nevertheless, it might be the complaint that brings the patient to the ophthalmologist, who should not dismiss it as an unimportant disturbance of accommodation but recognize it as a possible sign of a neurosis.

Stefan Van Wien.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

1

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Pau, H. and Conrads, H. **Contribution to the physiology and pathology of the smallest corneal nerves.** Arch. f. Ophth. 157:356-360, 1956.

The authors studied the endings of the corneal nerves and examined rabbits' corneas using the Gross-Schultze silver-stain method, modified by Stoehr. They feel that the cytoplasm of Schwann's cells and the parenchymal cells of the cornea together represent a transmitting plasmodium in which the nerve fibers are situated. After an injury, new Schwann cells arise from migrating corneal parenchymatous cells. The corneal cells may or may not represent trophic centers of nervous elements. (6 figures, 3 references) Ernst Schmerl.

Reiser, K. A. and Colmant, H. J. **Changes of the corneal nerves in keratoplasty. Experiments and histologic studies.** Arch. f. Ophth. 157:314-355, 1956.

For histologic studies of changes in the nerve fibers in corneal transplants and in the hosts' corneas the authors used 18 rabbits and stained the nervous tissue according to the method of Bielschowsky-Gross. Implants from 2 to 467 days old were examined. Two days after surgery the nerve fibers of the implanted tissue showed pronounced degeneration. Retrograde degeneration of nerve fibers of the hosts' corneas was slight. About one week after surgery regeneration of the hosts' nerve fibers became noticeable. Fibers and Schwann's cells arose from large nerve trunks and polinuclear corneal cells became connected with the regenerating nerve fibers. These so-called pre-nervous pathways showed smallest granula arranged in rows. Nerve fibers seemed to develop from this preformed tissue. Another type of regeneration was seen two to three weeks after surgery. The hosts' corneas showed nerve fibers arranged in geometric patterns. Regenerative innervation of the transplants started about 50 days after surgery. (18 figures, 42 references) Ernst Schmerl.

Rohen, Johannes. **Arteriovenous anastomoses in the limbus area of the dog.** Arch. f. Ophth. 157:361-367, 1956.

The author studied histologically the angle of the anterior chamber and its vascularization. He did not find a typical Schlemm's canal but large Fontana's

spaces and deep scleral venous plexuses. Numerous arteriovenous anastomoses were seen especially between branches of the anterior ciliary arteries and the large intrascleral veins. Epithelioid cells covered large areas of the walls of the anastomoses. The function of this arrangement of blood vessels is probably the regulation of the circulation of the blood and aqueous. (4 figures, 8 references)

Ernst Schmerl.

Tansley, Katharine. **Comparison of the lamina cribrosa in mammalian species with good and indifferent vision.** Brit. J. Ophth. **40**:178-182, March, 1956.

The lamina cribrosa is a network of collagen, elastic and neuroglial fibers, partly continuous with the sclera through which the optic fibers run on their way through the back of the eyeball. This helps to maintain the rigidity of the sclera at this otherwise weak point. In the mouse, the rat and the rabbit these fibers are very poorly developed while in cats, squirrels and monkeys the lamina cribrosa is quite well developed. (8 figures, 8 references) Morris Kaplan.

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Niedermeier, Siegfried. **Experimental investigations into the pathogenesis of papilledema.** Arch. f. Ophth. **157**:397-406, 1956.

The author produced papilledema in a number of rabbits by intradural injections of Periston followed by vital staining. Increase of intracranial pressure seems to affect the vascular permeability especially in the area of the lamina cribrosa. The sympathetic nervous system seems to be involved. However, papilledema does not seem to develop as part of a general edema, but as a local process. (4 figures, 18 references)

Ernst Schmerl.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Akiya, S. **Determination of phosphorous metabolism in the retina by radioactive P^{32}.** Acta Soc. Ophth. Japan **60**: 826-833, July, 1956.

Radioactive P^{32} in the form of a phosphate was injected into rabbits. The eyes were removed 24 hours after the injection. Fractions of phosphorous compounds were obtained from the retina by Schneider's and Schmidt-Tannhäuser's procedure, then the specific radioactivity was determined in each fraction. Radioactive phosphoprotein increased very much in the retina during the 24-hour period after the injection, next ribonucleic acid phosphorus and phospholipid. Radioactive deoxiribonucleic acid phosphorus is hardly demonstrable. The increase in the radioactive ribonucleic acid phosphorus and phosphoprotein is greater in light adaptation than in dark adaptation. An administration of KCN, dinitrophenol or NaF into rabbits prior to phosphorous administration impedes the increase of the phosphorous compounds in the retina. (4 tables, 17 references) Yukihiko Mitsui.

Cohan, Bruce E. **Aqueous humor outflow: an experimental study using radiopaque materials.** A.M.A. Arch. Ophth. **55**:792-799, June, 1956.

The author describes his technique of introducing radiopaque materials into the anterior chamber of rabbits' eyes. He illustrates the path of aqueous outflow from the anterior chamber of the rabbit through the ciliioscleral sinus to the aqueous veins. (5 figures, 18 references)

G. S. Tyner.

Erickson, O. F., Feeney, L. and McEwen, W. K. **Filter-paper electrophoresis of tears.** A.M.A. Arch. Ophth. **55**:800-806, June, 1956.

Using filter-paper electrophoresis, the author examined the tears of 20 different species of reptiles, birds, and mammals. Several species of animals have a "slow-moving lysozyme" component, but it has only about three-fourths of the electrophoretic mobility of egg-white and human lysozyme. All the other animal tears tested showed no lysozyme within the limits of the test. The tear fluid appears to be a true secretion. (1 figure, 1 table, 7 references) G. S. Tyner.

François, J. and Rabaey, M. **The proteins of the corneal epithelium.** Ann. d'ocul. **189**:445-458, May, 1956.

The authors' studies show the corneal epithelium of the ox to contain at least four water-soluble protein fractions, of which one predominates. None is a lipoprotein. One is P.A.S. positive and is probably either a muco- or glyco-protein or a glycogen-protein complex. (15 figures, 11 references) John C. Locke.

Kornblueth, W. and Ben-Shlomo, E. **Glucose utilization of the retina.** A.M.A. Arch. Ophth. **55**:813-817, June, 1956.

This report deals with the influence on the glucose uptake of the retina of some substances which cause toxic amblyopia and some compounds which damage the visual cells. Methyl alcohol, quinine, sodium cacodylate, formaldehyde, sodium iodate and ozide, physostigmine, isoflurophate (DFP), acetazoleamide (Diamox), ethionine, aminopterin and acidosis had no effect on the glucose utilization of the retina. Iodoacetic acid caused a 30 percent inhibition. (4 tables, 14 references) G. S. Tyner.

Kovalev, D. **The role of hyaluronic acid and hyaluronidase in the pathology of the visual organ.** Vestnik oftal. **4**:12-13, July-Aug., 1956.

Tests showed that hyaluronidase was present in the iris and ciliary body of 31 normal bulls' eyes and also in human eyes with various diseases. The iris and ciliary body of glaucomatous eyes either have no hyaluronidase activity or it is decreased. The author points out that there is a certain equilibrium between the hyaluronic acid of the vitreous and hyaluronidase of the iris and ciliary body which is regulated by the central nervous system. Inactivation (for unknown reasons) of hyaluronidase in glaucoma disturbs the equilibrium. Polymerization of hyaluronic acid is increased, the colloids of the vitreous absorbs more molecules of water, the vitreous swells, and as a result there is an increase of the intraocular pressure. The pressure also can be increased because of the slow outflow of the aqueous which contains the more viscous hyaluronic acid.

Olga Sitchevska.

Nachminovitch, I. **The permeability of the cornea and ways by which medicaments penetrate into the eye.** Vestnik oftal. **4**:3-9, July-Aug., 1956.

A review of the literature and of previous work suggests that the possibility of penetration of drugs into the anterior chamber by osmosis, diffusion or by biochemical absorption can be fully rejected. This seems improbable, as clinically the drugs do penetrate into the eye. The author, therefore, used the objective factor of time—chronaximetry, that is, the definition of the least intervals of time during which the action of the neurotropic substances used in ophthalmology (atropin, pilocarpin,) is manifested. Experiments were done on bulls' eyes, enucleated in two or three hours after killing of the animal. The technique of the experiments is described in detail. In human eyes (15 tests), drops of atropine or pilocarpine were instilled into the eye, the eyeball was immediately washed copiously with normal saline and the time was watched until the action of the drug took place.

The results of the two series of tests showed that the drugs penetrated the dead cornea after being in contact with the cornea for about eight hours and penetrated the living cornea after a few seconds. This speedy physiologic process is characteristic only of nerve elements. Thus, in introducing the alkaloids into the eye, their transposition occurs from the surface of the cornea, through its nervous apparatus, to the iris, possibly through the end nerves of the corneal tissue. This function of the nervous elements is named conditionally by the author "the molecular-kinetic property" of the nervous tissue, which needs further experimental work. Positive findings might throw more light on the trophic function of the nervous system and on the nutrition of avascular tissues.

Olga Sitchevska.

Renard, G. and Bouzas, A. **Tolerance and activity of isonicotinylhydrazine (I.N.H.) in ophthalmology.** Arch. d'opht. **16**:253-260, April-May, 1956.

The authors review the literature on the hydrazide of isonicotinic acid in the treatment of systemic and ocular tuberculosis. They refer particularly to the studies of Simonelli and Rizzini, confirmed by Bruna et al., which showed that the blood-aqueous barrier was permeable to I.N.H. and that the concentration of this substance in the aqueous was elevated seven times more after subconjunctival administration than after systemic. Renard and Bouzas studied the tolerance of the ocular tissues to instillations and injections of the drug. They found that instillations were well tolerated and that subconjunctival injections were well tolerated in daily dosages up to 50 mg. in the rabbit and up to 25 mg. in man. In the rabbit intracorneal injection was also well tolerated, as were injections into the anterior chamber in doses equal or inferior to 5 mg. Intravitreal injections, on the other hand, produced disorganization in doses of 5, 7.5, and 10 mg. Retrobulbar injections in doses of 5 to 25 mg. were well tolerated.

In experimental tuberculosis of the rabbit, I.N.H. by subconjunctival injection prevented the appearance of inflammatory lesions when administered soon after inoculation. The lesions healed in about a month when the drug was administered daily before the twenty-first day after inoculation. P. Thygeson.

Schweer, G. and Karell, E. **Glucosamine of the vitreous body. Third report.** Arch. f. Ophth. **157**:422-428, 1956.

The authors continued their studies and found that the vitreous fluid of beef is much richer in hyaluronic acid than the fibrils. The concentration of glucosamine in the posterior half of the vitreous body is much higher than in the anterior parts. It seems to be doubtful whether the ciliary body is a primary source of hyaluronic acid. (3 tables, 22 references)

Ernst Schmerl.

Takagi, Y. and Hama, H. **Distribution of β-glucuronidase in ocular tissues.** Acta Soc. Ophth. Japan **60**:653-658, July, 1956.

The β-glucuronidase is extracted from various tissues of animal eyes and the activity of this ferment is measured by Talalay-Fishman's method. In most animals the activity of this ferment is most vigorous in the uvea, particularly in the ciliary body, then in the retina and choroid. It is very weak in the lens, vitreous and serum. Among animals cats have the highest level of this ferment. This ferment increases in the secondary aqueous. When such agents as phenol, phenolphthalein and menthol are administered for four days before killing the animal, the activity of the ferment in the uvea is considerably suppressed, but that in the liver is not. (12 figures, 18 references)

Yukihiko Mitsui.

Wudka, E. and Leopold, I. H. **Experimental studies of the choroidal vessels.** A.M.A. Arch. Ophth. **55**:857-885, June, 1956.

The authors discuss the innervation of the choroid and the results of their study of the action of a large number of drugs on the vessels of this structure. The ciliary nerves supplying the choroid contain sympathetic, parasympathetic, and sensory fibers. This long, concentrated and informative report cannot be adequately abstracted. (11 figures, 5 tables, 142 references) G. S. Tyner.

Wyt, L. and Schwab, F. **Temperature changes in the vitreous after ultrasonic radiation.** Strahlentherapie **99**:600-609, 1956.

The authors show that exposure for one minute of the living rabbit's eye to ultrasound waves of intensities comparable to those used therapeutically raises the temperature of the vitreous significantly. Continuous application of sound with an intensity of 1.25 Watt/cm. gives rise to a temperature increase of 0.87° C. With impulse sound irradiation with sufficient tactile proportions (1:5, 1:10) even high intensities or amplitude peaks can be irradiated into tissue without essential increase of temperature. (2 figures, 6 tables, 13 references)

F. H. Haessler.

Yamaji, R. and Matsumoto, T. **Regeneration of visual purple, I. Effect of methionine.** Acta Soc. Ophth. Japan **60**: 833-837, July, 1956.

The regeneration of the visual purple is accelerated when methionine, homocysteine or cysteine is injected into frogs before killing the animals. Homocysteine and cysteine have a reverse action. Parachlormercuribenzoate also inhibits the regeneration of visual purple by itself and it also suppresses the accelerating effect of methionine. Both of these inhibitions are, however, removed by an addition of reduced glutathione. The authors suggest that the SH-groups in methionine play an important role in accelerating the regeneration of visual purple. (1 figure, 1 table, 8 references)

Yukihiko Mitsui.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Christoferson, K. W. and Ogle, K. N. **The effect of homatropine on the accommodation-convergence association.** A.M.A. Arch. Ophth. **55**:779-791, June, 1956.

The accommodative convergence is defined as that portion of the total convergence of the eyes that is initiated by the stimulus to accommodation by virtue of the accommodation-convergence synkinesis. Some think that the association is a labile one and is entirely learned, and others believe that the association of the two functions is innate and generally invariable. There are also those who combine these concepts.

Ten nonpresbyopic persons served as subjects for this study of the effect of homatropine. The authors conclude that the ratio of the accommodative convergence to an accommodation-stimulus change, the (A-C)/A ratio increases markedly with increasing degrees of cycloplegia produced by homatropine. They believe that the effect of age on the accommodative mechanism is fundamentally different from the effect of drugs which partially paralyze the ciliary muscle and where the (A-C)/A ratio remains more or less normal. (7 figures, 18 references) G. S. Tyner.

Gardiner, Peter A. **Observations on the food habits of myopic children.** Brit. Med. J. **2**:699-700, Sept. 22, 1956.

It was found that myopic children are more particular about their sources of

first-class protein than other children. Further study is needed to establish a definite correlation of these two data. (1 figure, 3 tables, 3 references)

Irwin E. Gaynon.

Hallden, Ulf. **An optical explanation of Hering-Hillebrand's horopter deviation.** A.M.A. Arch. Ophth. **55**:830-835, June, 1956.

An equation is presented to explain Hering-Hillebrand's horopter deviation, the validity of which is limited to the central part of the longitudinal horopter. (2 figures, 9 references) G. S. Tyner.

Miles, Paul W. **Optics and visual physiology.** A.M.A. Arch. Ophth. **55**:888-908, June, 1956.

The annual review of optics and visual physiology has been written in a very interesting and readable fashion. (187 references) G. S. Tyner.

Prince, J. H. and Fry, G. A. **The effect of errors of refraction on visual acuity.** Am. J. Optometry **33**:353-373, July, 1956.

The effect on visual acuity of various degrees of ametropia, induced by placing various ophthalmic lenses before the eyes, was tested on several subjects. The acuity was tested on the Landolt "C," which was presented in eight different directions in random order. Results were analyzed statistically. The tests were repeated for plus spheres and also for astigmatism from plus cylinders with or against the rule. Tests performed in the morning were compared with tests on the same subjects done in the evening to determine the possible effect of fatigue. Visual efficiency, if anything, tended to improve rather than diminish toward the end of the day. Paul W. Miles.

Remberg, Heinrich. **The Purkinje shift in redgreen blindness and its diagnostic meaning.** Arch. f. Ophth. **157**:412-421, 1956.

The author describes a photometric procedure which permits exact measurements of various states of adaptation in dim light. Details of methods and theory are discussed at length. (1 figure, 8 references) Ernst Schmerl.

Westheimer, G. and Mitchell, A. M. **Eye movement responses to convergence stimuli.** A.M.A. Arch. Ophth. **55**:848-856, June, 1956.

The experiments were carried out to ascertain the time characteristics of eye movements elicited by the presentation of visual stimuli in horizontal disparity. Photographic eye movement records were obtained showing responses to convergence and convergence relaxation stimuli. Convergence movements show a reaction time of about 0.15 to 0.20 second. A 1.5 degree convergence movement takes about 0.4 second to be completed, not including the reaction time. Higher levels of convergence take longer to reach. The final convergence level is often as much as a degree or so less than the stimulus without any reports of diplopia on the part of the subject. (7 figures, 8 references) G. S. Tyner.

5

DIAGNOSIS AND THERAPY

Barraquer-Moner, J. and Muinos Simon, A. **Lamellar scleral resection: the indications, technique, and results.** Arch. d'opht. **16**:154-164, 1956.

The authors describe the operation of scleral resection and mention that the original penetrating resection of Lindner, which was technically difficult and subject to complications, has been replaced, except in certain severe cases incurable by other means, by the lamellar technique. This operation, which is almost without danger, retains all the ad-

vantages of the Lindner procedure. Created for the treatment of retinal detachment, the operation has also been used with success in the treatment of severe myopia. The authors then review in detail the indications for the operation, mentioning particularly detachment in aphakic patients, in myopes, in cases of retraction of the vitreous, in large disinsertions, and in recurrences. In addition they advocate its use in high myopia with sclerochoroidal degeneration, and after the operation of Fukala to correct the residual astigmatism and to ameliorate the sclerochoroiditis. They describe the technique in detail, stress the importance of locating the resection as near the equator as possible where its effect will be greatest, and call attention to the necessity of avoiding the vortex veins. They report satisfactory improvement in 73 percent of cases of myopia, and cure of detachment in 82 percent.

P. Thygeson.

Bonnet, P. and Bret, P.-V. **Tomography of the intracranial carotid artery.** J. Med. Lyon, **37**:531-539, July 5, 1956.

During the past two years the authors have X rayed the skull of all patients with suspected atheromatous plaques of the carotid, particularly those with obvious atheromata of the retinal vessels, using the technique of tomography (planigraphy). This method gives a section through the carotid at a specific level with blurring of all the structures lying anterior and posterior to the plane desired. The authors feel that this gives them information that routine X rays omit and that it is a valuable adjunct to arteriography. (12 figures)

David Shoch.

van Boort, H. J. J., Warmoltz, N. and Winkelman, J. B. **Colour photography of the human retina with a discharge flashlamp.** Med. & Biol. Illustration **6**:166-170, July, 1956.

The authors describe a quartz discharge flashlamp which they used in the Zeiss Nordenson camera and obtained colored photographs of the fundus which are superior to those taken with other sources of light. (4 figures, 6 references)

F. H. Haessler.

Evans, P. Jameson. **Corneal needle—a modification.** Brit. J. Ophth. **40**:189, March, 1956.

The axis of the eye is antero-posterior rather than side to side. The suture is pulled through more easily with this new needle. (1 reference) Morris Kaplan.

Fronimopoulos, J. and Dimitrion, T. **Clinical and physiologic observations on the subconjunctival and retrobulbar injection of air.** Arch. d'opht. **16**:137-142, 1956.

The authors have found subconjunctival injection of air a very useful therapeutic agent to promote absorption of subconjunctival hemorrhages from different causes. No therapeutic effect was noted in inflammations of the anterior segment nor in hemorrhages of the posterior segment. Retrobulbar injections of air were without effect on hemorrhages of the fundus, but had some influence on ocular tonus. Immediately after injection a sharp increase in tonus of 10 to 15 mm. of Hg was noted. This increase was maintained for 20 to 60 minutes and was followed by a drop to between 2 and 5 mm. above the base pressure. This slight elevation was maintained for several days. The authors explain this tonus effect as a result of action on the ciliary ganglion in the course of which an initial inhibition of parasympathetic fibers is followed by an inhibition of sympathetic fibers.

P. Thygeson.

Lossius, H. M. **Transitory visual dis-**

turbances after ventriculography. Acta psychiat. et neurol. Scandinav. **31**:81-86, 1956.

Three cases of disturbance of vision as a direct result of ventriculography are reported. In all patients there was diminution of vision (to complete blindness in one) and a homonymous defect in the left lower quadrants. The disurbances are rare and transitory and are ascribed to a derangement of the vasomotor control of cerebral vessels. F. H. Haessler.

Mehta, K. H. **A new occluder.** Brit. J. Ophth. **40**:188, March, 1956.

A new occluder is briefly described which was patterned after the Doyne's occluder which adheres to the posterior surface of spectacle glass. It differs from the older one in that it is composed of soft, flesh-colored, plastic material. (4 figures) Morris Kaplan.

Noyori, S. and Tanaka, H. **A hand retinal camera. II.** Acta Soc. Ophth. Japan **60**:821-825, July, 1956.

A new hand retinal camera is described. It is based on the principle of the direct ophthalmoscope with monoflex camera and electric flash light. It is very handy but is still efficient. The exposure time of 1/500 second does not allow blurring of the picture by shaking, thus 90 percent of the exposures are successful. (10 figures, 1 reference)

Yukihiko Mitsui.

Sjögren, Henrik. **Keratoplasty with punch forceps.** Tr. Ophth. Soc. U. Kingdom **75**:321-325, 1955.

The author has had a punch forceps made that will give a graft and a hole of the same size with perpendicular sides. Both jaws move against each other. Punches of 3, 4 and 5 mm. diameter have been made by Stille of Stockholm, Sweden. (6 figures, 2 references)

Beulah Cushman.

Swan, Kenneth C. **Resection under Tenon's capsule.** A.M.A. Arch. Ophth. **55**:836-840, June, 1956.

This paper on the technique and advantages of resection under Tenon's capsule is a supplement to a previous one by the same author. (5 figures, 5 references)

G. S. Tyner.

6

OCULAR MOTILITY

Burian, Hermann M. **Treatment of amblyopia ex anopsia with eccentric fixation.** Iowa St. M. Soc. J. **46**:449-455, Aug., 1956.

Burian reports 42 cases of strabismus in 35 of which there was amblyopia with eccentric fixation and in seven amblyopia with central fixation. In spite of the classical teaching that occlusion of the "good" eye simply reinforces eccentric fixation in the amblyopic eye, Burian reports an almost uniform improvement in vision in the younger children and in those whose cooperation was good. He emphasizes that *complete* occlusion is necessary and that it must be continued even after 20/20 vision is obtained since there is some tendency to regression in an amblyopic eye. He presents graphic data on six patients; five treated successfully and one failure.

In a field where "clinical impressions" are rife and where there is very little impartial evaluation of results, this short paper is a model of restraint and objectivity. (6 figures, 9 references)

David Shoch.

Mannironi, R. and Toselli, C. **Clinical contribution to the study of recurrent and alternating external ophthalmoplegias.** Riv. oto-neuro-oftal. **33**:223-238, May-June, 1955.

The author describes a case of recurrent and alternating external ophthalmoplegia. Emphasis is placed on the electro-

encephalographic characteristics and their significance. (11 references)

William C. Caccamise.

de Muelenaere, H. **The nonsurgical treatment of concomitant strabismus at Westminster Eye Hospital, London.** Ann. d'ocul. **189**:402-415, April, 1956.

The methods in use at the Westminster Eye Hospital are described.

John C. Locke.

7

CONJUNCTIVA, CORNEA, SCLERA

Blaxter, P. L. **Conjunctival lymphoma treated by X ray.** Tr. Ophth. Soc. U. Kingdom **75**:315-320, 1955.

A 22-year-old woman with a family history of tuberculosis had a conjunctival swelling. A histologic diagnosis of lymphoma was made and the lesion disappeared after a single dose of 300 r at 130 kV within three weeks. (4 figures, 7 references)

Beulah Cushman.

Bonnet, P. **Corneal involvement in vernal catarrh.** Arch. d'opht. **16**:143-153, 1956.

The author notes the paucity of knowledge on the pathogenesis of vernal catarrh and the multiplicity of the clinical manifestations of the disease. He mentions the rarity of the condition in France, and that in 23 years in an active university clinic he saw personally only 50 cases. After reviewing the conjunctival features of the disease, of which papillary hypertrophy is the most prominent, Bonnet considers the corneal changes, stressing that the limbal and corneal changes differ essentially from the vascular pannus of trachoma. These changes he believes are papillary in type. He describes in detail the limbal and corneal changes in three cases and illustrates them with three plates in color. In one case there were cystic formations at the limbus suggesting by their position the limbal follicles of trachoma but easily differentiable from the latter by biomicroscopic examination.

P. Thygeson.

Greaves, D. P. **Bowen's disease of the cornea.** Tr. Ophth. Soc. U. Kingdom **75**: 311-314, 1955.

A woman, 81 years of age, was reported to have an intra-epithelioma of the cornea, after histologic examination. Another part of the limbus showed only epithelial hyperplasia. The lesion was treated with superficial application of X rays in daily doses of 400 r five days a week (total 5,000 r). (2 figures, 6 references)

Beulah Cushman.

Gurd, D. P. **Treatment of corneal infections due to Pseudomonas pyocyaneus with polymyxin B sulphate ointment.** Brit. J. Ophth. **40**:159-166, March, 1956.

In two cases of suspected but not bacteriologically proved pseudomonas infection of the cornea, the lesion disappeared after the application, four times daily, of an ointment containing 10,000 units of polymyxin B sulfate and 500 units of bacitracin per gram. In a controlled series of corneal infections in rabbits, treatment with the ointment gave very encouraging results. (12 figures, 3 references)

Morris Kaplan

Janert, H. **Precancerous conditions of the cornea and conjunctiva.** Arch. f. Ophth. **157**:380-396, 1956.

The author describes clinically and histologically a case of Bowen's disease of the corneae and two cases of erythroplasia of the conjunctiva. The patient with Bowen's disease had pronounced facial keratomas; this supports the view that Bowen's disease is a kind of dyskeratosis. Excision of the suspicious tissue seems to be indicated. (11 figures, 53 references)

Ernst Schmerl.

Jawetz, E., Thygeson, P., Hanna, L., Nicholas, A. and Kimura, S. **Antibodies to APC virus type 8 in epidemic keratoconjunctivitis.** Proc. Soc. Exper. Biol. & Med. **92**:91-95, May, 1956.

The sera from patients with epidemic keratoconjunctivitis in Japan, Italy, Switzerland, and North America contain neutralizing antibodies to APC virus type 8. Paired sera from such patients show a significant rise in neutralizing antibodies to APC virus type 8. These antibodies are present for a limited period after the onset of the disease. The constant association of epidemic keratoconjunctivitis and APC virus type 8 suggests that this agent, or an antigenically related virus is a factor in the etiology of this disease. (4 tables, 8 references)

Irwin E. Gaynon.

Pick, Walter. **Parinaud's oculoglandular syndrome.** J. Pediat., **49**:316-319, Sept., 1956.

The author presents a case of Parinaud's syndrome that is of unusual interest in that the presenting complaint of the patient was a preauricular mass. Attention was drawn to the eyes only because of an apparent squint which was actually the result of an edema of the upper lid. Response to oral tetracycline and local sulfathiazole was prompt. (2 figures, 12 references) David Shoch.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Boeke, Wilhelm. **Benign tumors of the iris.** Arch. f. Ophth. **157**:368-379, 1956.

The author studied a tumor of the iris. The eye had been removed because the tumor kept growing and malignancy had to be taken into consideration. The sections were stained with hematoxylin and eosine, van Gieson, Malory and Gomori stainings. The question arose whether the tumor is a leiomyoma or a neuroma. The author feels that a differential diagnosis can only be made when a thionin-tartaric acid inclusion staining is made. The demonstration of myoglia is not considered sufficient proof for the presence of a leiomyoma. Structural similarities in myogenic and neurogenic tumors of the iris cannot be explained by a common embryonic background. (5 figures, 28 references) Ernst Schmerl.

Goldberg, H. K. **Malignant melanoma of the iris: case report and brief review of the literature.** Sinai Hosp. J. **5**:83-89, April, 1956.

The author's study of an eye and a review of the literature suggests that demonstration by gonioscopy of invasion of the filtration angle by a tumor of the iris is an indication of malignancy and justifies immediate enucleation. In the patient described, mobility of one segment of the iris was impaired and there was evidence of a heavily pigmented tumor in the posterior part of the iris.

F. H. Haessler.

Offret, G. and Haye, C. **Results obtained in the treatment of uveitis by cortisone. (Five years' experience.)** Arch. d'opht. **16**:261-273, April-May, 1956.

The authors report the use of cortisone or hydrocortisone treatment in 150 cases of acute and chronic uveitis, of which 110 were chronic iridocyclitis, 20 were choroiditis, and 20 were acute iridocyclitis. In the 110 cases of chronic iridocyclitis, all but 36 were classified as manifestations of one of the uveitis syndromes or as having a presumptive cause. The hormones were used topically and in some instances systemically. The results of treatment are given in detail. The steroids proved to be very efficacious in acute iridocyclitis and caused a return to normal tension in 60 percent of the cases of uveitis with hypertension. Treatment was much less efficacious in the chronic forms

of uveitis, especially in uveitis associated with the menopause. The effect was poor in the cases of Fuchs's syndrome and in certain cases of iridocyclitis of rheumatic origin. Results were only fair in uveitis believed to be due to tuberculous allergy or to focal infection. Best results were obtained in post-traumatic uveitis, in iridocyclitis accompanying interstitial keratitis, and in sympathetic ophthalmia. There were unfortunately a number of cases of uveitis which responded brilliantly to the hormones, only to relapse sharply when their use was discontinued.

P. Thygeson.

Yoshida, E. **Experimental studies on the pathogenesis of idiopathic uveitis.** Acta Soc. Ophth. Japan **60**:642-653, July, 1956.

The allantoic fluid from chick embryos older than 13 days of incubation causes a uveitis in rabbits when injected into the vitreous. The uveitis is transferable when the vitreous of the inflamed eye is injected intravenously into other rabbits. The allantoic fluid of younger embryos does not cause such a uveitis. The allantoic fluid of old chick embryos is antigenic; and this fact is proved by the author by the precipitation reaction. (12 tables, 26 references)

Yukihiko Mitsui.

Zarrabi, M. and Parhami, Y. **A case of Still's disease with ocular complications.** Arch. d'opht. **16**:177-179, 1956.

The authors report the case of an eight-year-old girl with Still's disease commencing at the age of six years. Simultaneously with the onset of the joint disease a bilateral iridocyclitis occurred which became chronic and was complicated by band-shaped keratopathy. Systemically, in addition to the arthritis, there was fever, tachycardia, enlargement of the liver and spleen, malnutrition, and protrusion of the abdomen. Cortisone was given systemically and topically in the eyes with spectacular amelioration which has persisted. Cortisone has been instilled into the conjunctival sac once daily since the beginning of treatment. Although the liver and spleen have remained enlarged, the child has been able to return to normal activity.

P. Thygeson.

9

GLAUCOMA AND OCULAR TENSION

Baron, J. B., Payrau, P. and Bessineton, J. C. **Nocturnal variations in the organic and inorganic ion content of the serum in patients with chronic ocular hypertension (also variations under the influence of antithyroid drugs).** Bull. et mém. Soc. franç. d'opht. **68**:492-497, May, 1955.

Variations in the quantities of organic and inorganic ions in the serum of patients with chronic glaucoma were established. These variations could be caused by light, darkness, and provoked by antithyroid drugs. Two main groups were distinguished: in the first group inorganic and organic ions showed variations, globulins disappeared, β globulins increased and the ocular tension rose in reduced illumination, and antithyroid drugs were ineffective; in the second group only the organic ions showed variations, darkness caused a decrease in α, β, and γ globulins and raised the ocular tension, antithyroid drugs induced a return of the ion content of the serum to normal values and reduced the ocular tension. (3 tables)

Alice R. Deutsch.

Cambiaggi, A., Spurgeon, W. M. and Spurgeon, R. **Diurnal changes in eosinophile count.** A.M.A. Arch. Ophth. **55**:765-778, June, 1956.

In these experiments 35 glaucomatous and 14 normal eyes were used. In about one quarter of the cases of chronic simple glaucoma there was a high correla-

tion between the diurnal variations of the intraocular pressure and of the number of circulating eosinophiles. None of the normal eyes showed a relation between the appearance of the aqueous veins, on the one hand, and the eosinophile count and intraocular pressure, on the other; however, such a relation was found in six of the 30 glaucomatous eyes in which the aqueous veins were studied. (2 figures, 10 tables, 33 references)

G. S. Tyner.

Fukumoto, H. **The influence of autonomic drugs upon ocular tension. II and III.** Acta Soc. Ophth. Japan **60**:157-163, and 357-363, March and May, 1956.

This is a study of eyeball compression tests after administration of some autonomic drugs. After an instillation of homatropine or epinephrine in normal eyes, compression of the eyeball produces a reduction in the tension. After pilocarpine there is no such reduction in the tension, but there seems to be a delay in the recovery of the original tension after the compression.

Fukumoto further studied the effect of the combined administration of several drugs to cause a simultaneous blocking of both of the autonomic nerves or to cause a stimulation of one nerve with a blocking of another. He concludes that by a sympathetic stimulation, the suppression of aqueous production overwhelms that of the aqueous outflow to result in a greater lowering of ocular tension by eyeball compression. By a parasympathetic stimulation, acceleration of the aqueous production overwhelms that of aqueous outflow to result in a smaller lowering of tension by compression. (7 figures, 4 tables, 38 references)

Yukihiko Mitsui.

Gittler, R. and Pillat, B. **The use of BC 48 in the therapy of glaucoma.** Arch. f. Ophth. **157**:473-494, 1956.

BC 48 is decamethylen-bis-(N-methylcarbaminonyl - m - trimethyl - ammoniumphenol). In 67 eyes of 53 patients with glaucoma it was found to be as good a miotic as DFP and one which is easily soluble in water and does not deteriorate rapidly. In two-thirds of the patients a satisfactory reduction of tension was achieved, among them some who had been given other miotics without effect. (3 figures, 1 table, 22 references)

F. H. Haessler.

Gormaz, A. **Ridley's operation: late follow-up on two cases.** Arch. chil. de oftal. **12**:144-146, Aug.-Dec., 1955.

The author presents two cases of implantation of an acrylic lenticulus. In one of them, several months after surgery, the patient had several attacks of iritis and the lenticulus was luxated nasally into the anterior chamber. It had to be removed two years after its original insertion. In his second patient gonioscopic studies in several episodes of acute rise in tension revealed extensive anterior peripheral synechiae. The glaucoma was finally relieved by cyclodyalysis and a peripheral iridectomy. Walter Mayer.

Heinsius, E. and Maass, K. **Glaucoma operation with blunt and sharp diathermy.** Klin. Monatsbl. f. Augenh. **128**: 414-425, 1956.

One row of diathermy application is made 6 to 8 mm. from the limbus with a ball electrode (70-90 m A for 5 to 8 seconds). In addition about four perforating applications (2 mm. needle) are made in the same area, for escape of vitreous. 99 eyes were operated on and out of 67 eyes with chronic simple glaucoma 49 could be stabilized. The operation is advised when a fistulating operation is too dangerous, or in hemorrhagic and secondary glaucoma. (5 figures, 4 tables, 27 references)

Frederick C. Blodi.

Huerkamp, B. **Evaluation of intraocular pressure curves.** Klin. Monatsbl. f. Augenh. **128**:394-400, 1956.

It is emphasized that mean values (for a day or week) are more important than the peak values. The amplitude between the lowest and the highest reading increases in glaucoma and then the highest readings are often in the evening. The diurnal curve may become inverse. (8 figures, 2 tables, 11 references)

Frederick C. Blodi.

Kapuscinski, Witold Juliusz. **Clinical value of iridencleisis and cyclodiathermy as operations in lower intraocular pressure.** Klinika Oczna **25**:85-96, 1955.

The latter is the only bloodless operation, but to be successful requires precision in dosage. For cyclodiathermy the round tip is used at 7 mm. from the limbus, 12 to 15 applications of 15 seconds each are made. The tension was lowered in 22 of 26 eyes. Transient increase in pressure during the operation was seen in two cases. Iridencleisis is considered better than the Elliot and Lagrange operations. It was successful in 26 of 28 cases. Occasionally the author excised one of the iris pillars leaving the other one for the iridencleisis. (2 tables, 39 references)

Sylvan Brandon.

Kelly, T. Stuart-Black. **Some results of the Preziosi operation.** Brit. J. Ophth. **40**:222-224, April, 1956.

A trephine operation is described which employs the cautery to make the trephine hole. The report covers a three-year survey and 46 eyes. Of the eyes operated upon for chronic simple glaucoma, 93 percent developed normal tension (25 mg. or below). The Preziosis operation was described in 1924 but was promptly discarded; mention of this cautery method of surgery is difficult to find today. The results in this series justify the review of the procedure. The anesthesia can be similar to that ordinarily employed for glaucoma surgery. A conjunctival flap is prepared as in an iridencleisis. An ordinary glavanocautery is used at red heat and is passed through the limbus into the angle by one or more sweeps, similar to the ab externo incision. The escape of aqueous cools the cautery and helps avoid damage to the underlying structures. A pointed cautery is advised and the puncture hole should be large enough to admit an iris repositor. This puncture may be enlarged with the cautery or scissors. If the iris is burned it is of no consequence. Should it prolapse into the wound, it is perforated with the cautery and then reposited. A variation of the method is to bend the cautery point to the angle of a keratome and then use it as a keratome. If the iris does not budge into the hole, the cautery may be withdrawn and an iridectomy or inclusion operation performed at this point. Closure is by the usual method, with atropine and an antibiotic ointment and patch to conclude the procedure. Reaction is minimal and the patient is allowed up in 24 hours. Complications were few. There was one case of lens injury due to misdirection of the keratome. With the ab externo approach thermal cataract would be avoided. Successful drainage is achieved because a hard ring of scar tissue is produced, which is a barrier to fibroplasia. The results in secondary glaucomas were poor in the few hemorrhagic cases cited, but good in those due to uveitis. (4 references)

Lawrence L. Garner.

Langham, M. E. and Lee, P. M. **Action of the carbonic anhydrase inhibitor diamox on the formation of the aqueous humour.** Tr. Ophth. Soc. U. Kingdom **75**:229-243, 1955.

It was concluded that diamox reduces the rate of flow of the aqueous humor and disturbs the normal distribution of

bicarbonate between the blood and the aqueous humor and the possible causes of these changes are discussed. (4 figures, 2 tables, 12 references)

Beulah Cushman.

Martins Rocha, J. **Etiology of secondary glaucoma.** Rev. brasil. oftal. **15**:153-161, June, 1956.

The author very briefly reviews the different types of secondary glaucoma; in cases of inflammation, the secondary glaucoma may be produced during the inflammation or after it. He mentions glaucoma due to contusion and due to penetrating injury, glaucoma secondary to alterations in the shape of the lens such as microphakia or luxation of the lens, glaucoma secondary to iris atrophy, to alterations in the retinal vascular system, to retinal detachments and to intraocular hemorrhage. Walter Mayer.

Rhyne, J. L., Usar, M. C. and Carriker, F. R. **Idiopathic hypoparathyroidism and bilateral congenital glaucoma in the neonatal period.** Pediatrics **18**:448-454, Sept., 1956.

This is evidently the first reported case of idiopathic hypoparathyroidism in a newborn. The bilateral glaucoma appeared at the age of three months with steamy enlarged corneas, some proptosis, and tension of 50 in each eye. From the eleventh day, the infant had received large doses of vitamin D, phenobarbital (for convulsions), antibiotics, and a calcium-rich diet. Gonioscopy was negative. Miotics and diamox seemed ineffective, so several goniopunctures, goniotomy, and cyclodialysis were done. The glaucoma was considered to be unrelated to the general disease. (1 figure, 1 table, 16 references) Paul W. Miles.

Smith, Redmond. **Concerning glaucoma and retinal venous occlusion.** Tr. Ophth. Soc. U. Kingdom **75**:265-279, 1955.

Occlusion of the central vein may cause rubeosis iridis, and other conditions such as diabetic retinopathy, Eales's disease, retinal detachment and glaucoma may also produce rubeosis iridis. The relation of central vein occlusion as a complication of glaucoma, or whether the basic fault lies in an anatomic anomaly in the configuration of the angle of the anterior chamber is discussed, with the presentation of six cases and three pathologic specimens. In the material studied it would seem that thrombotic glaucoma was superimposed on primary glaucoma; a glaucoma was present in the other eye. (8 figures, 13 references)

Beulah Cushman.

Sourdille, G. P., Legrand, J. and Chevannes, H. **Synergism of diamox and surgery.** Tr. Ophth. Soc. U. Kingdom **75**: 245-247, 1955.

Diamox was effective in lowering intraocular pressure in most primary or secondary glaucoma. Pressure began to fall in half an hour, the action was reversible and control of the pressure was short-lived. The optimal dose was 250 mg. twice a day. Some patients complained of nausea and anorexia. The synergism of myotic drugs and diamox was impressive. Beulah Cushman.

10

CRYSTALLINE LENS

Duthie, O. M. **Bilateral cataract extraction.** Tr. Ophth. Soc. U. Kingdom **75**:25-31, 1955.

Bilateral intracapsular cataract extraction with its many advantages becomes a necessary consideration in order to preserve binocular single vision in younger patients. Beulah Cushman.

Gilkes, M. **Cataracts associated with local and general neurodermatitis.** Tr. Ophth. Soc. U. Kingdom **75**:301-310, 1955.

Two patients with cataract and general neurodermatitis associated with stress phenomena are reported. The neurodermatitis increased as the surgery was contemplated and cleared up after the operations were performed without previous warning. Each patient recovered 6/6 corrected vision. (8 figures)

Beulah Cushman.

Katorgina, O. **The classification of senile incipient cataract.** Vestnik oftal. **4**:30-35, July-Aug., 1956.

The author examined 102 eyes with incipient forms of cataract and classified them clinically as limited, advanced and mixed. The anatomical division was into cortical, nuclear and subcapsular cataract. The appearance of these forms of cataract as examined with the slitlamp and focal illumination is described. In the majority of the patients there was cortical cataract. (9 figures, 1 table) Olga Sitchevska.

Palin, A. and Tudway, R. C. **The use of radioactive isotopes in the diagnosis of intra-ocular malignancy.** Tr. Ophth. Soc. U. Kingdom **75**:281-299, 1955.

The authors studied the value of P^{32} in the diagnosis of tumor. Of the 862 eyes received at the Institute of Ophthalmology, which had been enucleated because of supposed intraocular malignant neoplasm, 12.9 percent were found to have no malignant growth. They found that the test in its present state is not to be regarded as a substitute for painstaking clinical judgment, but that it does have undoubted value as an adjunct where clinical consultant opinions are not unanimous. (13 figures, 14 references)

Beulah Cushman.

Reed, Howard. **Some observations on the visual results of cataract extractions.** Canad. M.A.J. **75**:113-115, July 15, 1956.

Between 1910 and now, cataract extraction in patients with a visual acuity of 20/30 or better, rose from 40 percent to 90. This was made possible by the acceptance of the intracapsular extraction. In the author's cases, delayed anterior chamber formation and hyphema was more common in the keratome incision group, although the visual acuity was not affected. The use of a Graefe knife or keratome is a matter of personal preference. Small variations in the technique of the number and types of preplaced or postplaced sutures do not materially influence the visual results. (5 tables, 14 references)

Irwin E. Gaynon.

11

RETINA AND VITREOUS

Fischer, Franz. **The newer therapeutic measures for diabetic retinopathy.** Therap. d. Gegenw. **95**:304-308, Aug., 1956.

The author gives a condensed account with brief but extensive bibliographic documentation of seven therapeutic measures, namely: 1. Rutin (vitamin P substances), 2. melanophore hormone and lipocaic hormone, 3. high protein diet, 4. vitamin E (tocopherol), 5. X-ray and radium therapy, 6. sexual hormone (androgens and estrogens), and 7. hypophysectomy and adrenalectomy. Rutin is as good as useless therapeutically although it has not been shown as yet that it may not prevent or postpone the development of retinopathy in a diabetic. In any case it does reduce capillary fragility. Experience with measures 2, 3, 4, and 5 does not justify any conclusions. Experience with sexual hormones is more extensive and more promising, but also not adequate as a basis of judgment. Measures 7 are still completely problematic. In short, in the absence of effective therapy one must concentrate on prevention for which the Joslin school has shown the way.

F. H. Haessler.

Lijo Pavia, J. **Internal exudative retinitis, treated successfully with hydrocortisone.** Rev. oto-neuro-oftal. **31**:42-46, April-May, 1956.

The author refers to a previously published paper in which he called attention to the fact that internal exudative retinitis is not often diagnosed. He feels that it is a definite disease entity mainly in young patients, without any definite etiology, which affects the innermost layers of the retina and is always reversible.

The author treated one of his patients with six subconjunctival injections of 12.5 mg. of hydrocortisone and the visual acuity improved considerably. Photoretinograms made before and after the treatment emphasize the fact that hydrocortisone can reverse the ophthalmoscopic findings of the disease. (2 figures, 10 references)

Walter Mayer.

Lijo Pavia, J. **Retinitis proliferans.** Rev. oto-neuro-oftal. **31**:37-41, April-May, 1956.

The author discusses briefly the pathology of this disease, the various theories of its origin, and also the ophthalmoscopic findings. Panoramic photoretinograms are of the greatest help for a differential diagnosis. He presents three cases of this disease with short case histories. (4 figures)

Walter Mayer.

Marinho de Queiroz, J. **Coats' retinitis and xanthomatosis.** Rev. brasil. oftal. **15**:137-150, June, 1956.

The author made a detained pathologic study of an eye with Coats' disease and feels that the foam cells, hemorrhages, granulomatous tissue, crystals of cholesterol, infiltration with round cells and the tendency to fibrosis are totally similar to the pathologic features of the xanthomatosis of the normocholesterinic group. The author feels that into this group belong the eosinophilic granuloma of the orbit, the Schueller-Hand Christian disease and gargoilism. He is now feeding cholesterol to rabbits experimentally, in an attempt to reproduce the same pathologic findings. (6 figures, 20 references)

Walter Mayer.

Marinho de Queiroz, J. **Serous choriopathy and retinopathy.** Rev. Brasil oftal. **15**:171-176, June, 1956.

The author studied three cadaver eyes used for keratoplasty and found localized edema of the retina and surrounding structures in different phases in all of them, which, the author believes, are probably manifestations of different stages of a serous choriopathy or retinopathy. (3 figures, 5 references) Walter Mayer.

McGaughey, R. S., Alexander, C. and Morrish, J. A. **The Groenblad-Strandberg syndrome. A report of three cases with massive gastrointestinal hemorrhage during pregnancy.** Gastroenterology **31**:156-168, August 1956.

The authors add three cases of massive gastrointestinal hemorrhage in patients with the Groenblad-Strandberg syndrome (pseudo-xanthoma elasticum, angioid streaks, and visceral manifestations) to the 11 cases which have been reported. These three cases are the first reported instances of massive gastrointestinal hemorrhage occurring in pregnant women who have the disease. The hemorrhage occurred only during pregnancy in these women and the relationship seems to be more than coincidental. Why pregnancy has an adverse effect on women with this disease is unknown. The disease should be suspected when there is upper gastrointestinal hemorrhage of obscure origin and the presence of pseudozanthoma elasticum, and angioid streaks of the retina should suggest this diagnosis at the bedside. (5 figures, 24 references)

F. H. Haessler.

Morgan, O. Gayer. **Some aspects of thrombosis of the retinal veins and its treatment.** Tr. Ophth. Soc. U. Kingdom **75**:3-24, 1955.

The author describes the pathogenesis and unique pathology of venous thrombosis. He gives practical evidence of the value of anticoagulants if the treatment can be initiated before the tissues have been torn by extravasated blood and that successful prophylaxis depends on anticipation of the actual thrombotic incident. (3 figures, 3 tables, 16 references)

Beulah Cushman.

Ōoka, R. and Ōgamï, T. **Effect of cold pressor test on retinal arterial pressure and its relation to the reaction in the central vascular system.** Acta Soc. Ophth. Japan **60**:740-745, July, 1956.

The blood pressure of the central retinal artery rises in the cold pressor test. However, the rise of the pressure in the retinal artery comes later than in the brachial artery. The recovery of the original tension after the test is also later in the retinal artery. It is also supposed that the reactivity of the retinal artery to the cold pressor test is smaller than that of the cerebral vascular system to an inhalation of CO_2. (4 figures, 2 tables, 9 references)

Yukihiko Mitsui.

Takeuchi, F. **A study of essential hypertension by measurement of the caliber of retinal blood vessels.** Acta Soc. Ophth. Japan **60**:745-754, July, 1956.

The measurement of the caliber of the retinal blood vessels is made with Mikuni's apparatus, which is a micro-scale attached to a binocular ophthalmoscope. A narrowing of the retinal vessel results with an advance of hypertension, particularly in the artery; thus a decrease in the ratio of the arterial caliber to the venous caliber results. A definite narrowing is observable in stages III and IV of Keith-Wagener and this fact may suggest that the narrowing of the artery in these stages is not only due to the sclerosis but also to a spasm of the artery. In contrast to Badtke, there is no dilatation of the artery in the earliest stage of hypertension. (1 figure, 8 tables, 14 references)

Yukihiko Mitsui.

13

NEURO-OPHTHALMOLOGY

Bonnet, Paul. **Ophthalmologic signs of cerebral tumors.** J. Med. Lyon **37**:511-530, July 5, 1956.

This is a rather comprehensive article which emphasizes the importance of a careful and complete ophthalmologic examination in every case of suspected intracranial abnormality. The author discusses papilledema at length and emphasizes quite properly that a careful study of the visual fields is far more important than the presence of papilledema in diagnosing and localizing cerebral tumors. Lesions of the optic pathways are presented in the classical anterior-posterior direction and a description of the visual field loss in each location given. Finally Bonnet presents the implications of various ocular palsies. This article will be of interest chiefly to French speaking ophthalmologists without access to Traquair or Walsh. (9 figures)

David Shoch.

Lein, J. N., Stewart, T. and Boll, F. C. **Sex-linked hereditary nystagmus.** Pediatrics **18**:214-217, Aug., 1956.

The authors present two pedigrees of families showing a hereditary sex-linked nystagmus. Apparently only four such pedigrees have been presented before. The defect occurred only in males but was transmitted by females. In every case the onset was at birth without any associated ocular disease. No other neurologic defects could be elicited. Color vision was normal but there was a diminution of cen-

tral vision in each case, apparently secondary to the nystagmus.

David Shoch.

Petersen, Peter. **Pupillography and the pupillogram.** Acta physiol. Scandinav. **37**:1-141, 1956.

This monograph cannot be abstracted adequately but we call attention to it here because it appeared in a periodic publication.

14

EYEBALL, ORBIT, SINUSES

Brain, Russell. **Exophthalmos.** Tr. Ophth. Soc. U. Kingdom **75**:351-356, 1955.

The exophthalmos and ophthalmoplegia are due to the swelling of the ocular muscles but occasionally the ophthalmoplegia may be apparent without exophthalmos. No clear clinical distinction exists between the ocular signs in thyrotoxicosis and in exophthalmic ophthalmoplegia, but the pathological changes are different. The author feels that Graves' disease and exophthalmic ophthalmoplegia are fundamentally alike—a hypothalamic-pituitary disturbance—and the difference is due to the degree of responsive activity of the thyroid and the effect on the other endocrine glands themselves and in the reactions of the peripheral tissues.

Exophthalmos in exophthalmic ophthalmoplegia is associated with resistance to backward displacement of the globe, unilateral or bilateral, some widening of the palpebral fissure, and lagging of the eyelid on looking downwards. Early conjunctival changes consist of edema, glistening, rolling easily into folds and increased vascularity. Elevation of the eyes is the movement affected first, then abduction. In extreme cases practically all movement can be lost.

Thyroidectomy is the treatment of choice in the thyrotoxic ophthalmoplegia, but if the exophthalmos is secondary to a pituitary-adrenal dysfunction thyroidectomy will make it worse. Treatment of the thyrotrophic exophthalmos at present seems to be X-ray radiation of either the pituitary or the orbits. (5 references)

Beulah Cushman.

Folken, G. G. **Posttraumatic intraocular hemorrhage.** Minnesota Med. **39**:440-443, July, 1956.

Hyphemia results from tears at the root of the iris, the ciliary body, or in Schlemm's canal. Vitreous hemorrhages follow rupture of blood vessels of the ciliary body, choroid, and at times, the retina. Secondary hemorrhages usually occur between the second and fourth day; secondary glaucoma is common at this time, the prognosis becomes poorer, and a simple paracentesis is indicated.

The patients should be kept in bed for one week with both eyes bandaged for a minimum of four days. The tension should be watched, and supportive treatment given. The use of a miotic is said to prevent secondary hemorrhage because the iris vessels are put on a stretch, the blood is squeezed out of the contracted ciliary muscle; and at the same time the canal of Schlemm should be opened to aid the absorption of blood in the anterior chamber. (14 references)

Irwin E. Gaynon.

Greene, Raymond. **Exophthalmos.** Tr. Ophth. Soc. U. Kingdom **75**:341-350, 1955.

The author in a review of the literature outlines the relation of exophthalmos to the thyroid and the exophthalmos-producing substance of the pituitary. Experimentally the exophthalmos of Graves's disease is associated with the influence of a substance or substances contained in pituitary extracts. The thyrotropic extract has been found to contain three factors, one of which raises the basal metabolic rate, another produces epithelial hyperplasia in the thyroid without exophthal-

mos and the third produces only exophthalmos. Thyrotophin which is second in its purest form had less effect on the eyes.

The exophthalmos-producing substance of the pituitary has been found in serum of exophthalmic patients. It and thyrotropine, though not identical, must be closely related. It has been found that corticotrophin with small doses of pituitary extract will produce exophthalmos in rats. It has also been noted that the thyroid stimulating factor of the thyrotrophic extract is inhibited by iodination, whereas the exophthalmos producing factor is not.

Endocrine disorders should be regarded as perversions of physiology rather than diseases. The author points out that we must study the edema and water content of tissues associated with the mucopolysaccharides and an increase in hyaluronic acid. It is most unlikely that a hormone causes the eyes to protrude. We must study the basic mechanisms of polysaccharide and fat metabolism and the manner in which they are effected by the body's hormones. (39 references)

Beulah Cushman.

Marinho de Queiroz, J. and Barbi, C. **Eosinophilic granuloma of the orbit.** Rev. brasil. oftal. **15**:163-168, June, 1956.

The authors describe a case of proven eosinophilic granuloma of the orbit which was cured by excission and curettage. They emphasize the importance of numerous X-ray films taken at different angles, which will help greatly in making a diagnosis if the most minute bony erosion is evaluated in these different views. Although it was not done in their case, the authors feel that X-ray treatment should be instituted after the surgical treatment. (3 figures, 9 references) Walter Mayer.

Pennybacker, J. **Exophthalmic ophthalmoplegia.** Tr. Ophth. Soc. U. Kingdom **75**:357-363, 1955.

The author discusses the treatment of 38 patients who were observed since 1938 and the effects of various treatment on the exophthalmos. The most extreme symptoms developed during the years of war stress; the affection is less common in periods of relative tranquillity. Therapy should consist of local treatment to lids and to the pituitary, and general endocrine treatment. Beulah Cushman.

Psenner, L. **Roentgenologic manifestations of space-taking lesions of the orbit.** Fortschr. a. d. Geb. d. Roentgenstrahlen **85**:125-141, Aug., 1956.

The author analyzes the general and particularly the roentgenologic diagnosis of the causes of exophthalmos in a number of illustrative cases. The reproductions of the X-ray films in question are excellent and truly illustrative. The commonest causes of exophthalmos are neoplasm and inflammation and the processes need not originate only in the soft and bony tissues of the orbit itself but in neighboring sinuses, the anterior and middle intracranial fossa and the epipharynx. (26 figures)

F. H. Haessler.

Stallard, H. B. **Exophthalmic ophthalmoplegia.** Tr. Ophth. Soc. U. Kingdom **75**:365-378, 1955.

Surgical intervention is indicated to insure immediate adequate and sustained covering and protection of the cornea, to reduce orbital edema and to relieve the damaging effects of compression and stretching of the intraorbital structures before the X-ray and medical treatment relieves the active phase of the thyrotropic disorder. Any ocular muscle surgery which may become necessary can be dealt with at the appropriate time. When progressive exophthalmos does not respond to medical treatment, a carefully performed tarsorrhaphy may hold and protect until radiation therapy has checked and reduced the exophthalmos. Small round

cells, which infiltrate the extraocular muscles, the orbital fat and connective tissue, are radio-sensitive, and treatment consists of 50 to 150 r on alternate days up to a dose of 800 r for four to six weeks. Recession of the globe usually begins within ten days and continues for five to eight weeks.

The effect of bony decompression according to Naffziger is often meager and does not relieve epiphora, chemosis or peri-orbital edema. (5 figures)

Beulah Cushman.

15

EYELIDS, LACRIMAL APPARATUS

Law, Frank W. **Dickey's operation for ptosis.** Tr. Ophth. Soc. U. Kingdom **75**:249-257, 1955.

The ideal operation for ptosis is one of shortening or advancing the levator tendon. The superior rectus should be used only when the levator has no function. The lid should only be attached to the frontalis muscle when there is no movement of the lid or globe. The Dickey operation is indicated when there is no movement upward of the lid or the globe, which produces progressively more elevation of the lid with single vision. (3 figures)

Beulah Cushman.

17

INJURIES

Arnold, E. L. **Gas gangrene panophthalmitis due to Cl. welchii.** Tr. Ophth. Soc. U. Kingdom **75**:259-263, 1955.

Brownish exudate in the anterior chamber and a quantity of pus of the same color was seen in the eye of a five-year-old boy, whose eye was removed after a penetrating wound of the cornea. A gram-positive bacillus identified as Cl. welchii (perfringens) which was sensitive to penicillin and aureomycin was found.

The absence of gas was noted in the clinical examination although a large amount was produced in the cultures. It is suggested that in all intraocular injuries the investigation include a search for anaerobic organisms. (9 references)

Beulah Cushman.

Reynon, M. and Belaïsch, A. **Traumatic arteriovenous caroticocavernous aneurysm.** Ann. d'ocul. **189**:365-379, April, 1956.

A case of caroticocavernous aneurysm with fatal termination is presented. Diagnosis had been confirmed by arteriography. Ligation of the common carotid artery had failed to control the ocular and general complications, and evisceration of the ipsilateral eye was required for neuroparalytic keratitis and glaucoma. Intracranial exploration revealed a dilated ophthalmic vein, compressing the optic nerve. Both were sectioned, and clips were placed on the intracranial portion of the internal carotid artery. The patient died a few hours later. The authors caution against the intracranial approach in other such cases. (4 figures, 15 references)

John C. Locke.

18

SYSTEMIC DISEASE AND PARASITES

François, J. and Verriest, G. **Syphilitic gumma of the hypophysis.** Ann. d'ocul. **189**:416-424, April, 1956.

A 38-year-old syphilitic woman developed headache, bitemporal hemianopia, and signs of pituitary hypofunction, near the end of her first pregnancy. After antiluetic therapy, the central vision returned to normal and the visual fields almost to normal. Diagnoses of functional enlargement of the pituitary gland, optochiasmal arachnoiditis and retrobulbar chiasmal neuritis were discarded in favor of a diagnosis of syphilitic gumma of the hypophysis. (5 figures, 7 references)

John C. Locke.

Georgiadès, G. **Fuchs' syndrome, the**

Barré-Liéou syndrome, and lesions of the cervical spine. Ann. d'ocul. **189**:380-391, April, 1956.

Abnormalities of the cervical spine, similar to those found in the Barré-Liéou syndrome, were demonstrated radiologically in 23 of 40 patients with Fuchs' heterochromic cyclitis. These included cervical rigidity, spondylolithiasis, increase or decrease of intervertebral spaces, osteophytosis, and decalcification. The fifth and sixth cervical vertebrae were the commonest sites for the lesions, and the first three cervical vertebrae were generally normal. The Barré-Liéou syndrome (retrobulbar pain, headache, tinnitus and vertigo) coexisted in 11 out of 55 cases. Radiologic study showed cervical lesions were present in seven out of ten cases. (6 figures, 15 references) John C. Locke.

Jawetz, E., Hanna, L., Kimura, S. J. and Thygeson, P. **Clinical diseases of adults associated with sporadic infections by APC viruses.** Arch. Int. Med. **98**:71-79, July, 1956.

Adenoidal - pharyngeal - conjunctival (APC) viruses are widely accepted as important etiologic agents in respiratory disease in military men and in children. It is becoming clear that a wide range of diseases, from minimal to severe, and involving one organ system or many, can occur. The majority of proved sporadic APC virus infections in adults observed by the authors have involved the eye. The constancy of eye affection has suggested that the eye may serve as portal of entry and this concept is supported by the difficulty of infecting volunteers with APC virus through the respiratory tract and the ease with which infection follows application of the virus to the conjunctiva. Cases have been observed in which conjunctivitis was the first symptom of disease and preceded systemic manifestations by several days. (3 figures, 14 references) Irwin E. Gaynon.

Ophthalmic Miniature

After an incision through the cornea, the retraction of the divided tissue is most marked at its anterior and posterior surfaces, so that when the lips of the wound come together again there is contact of the central areas of the substantia propria and wedge-shaped gaping spaces left anteriorly and posteriorly.

Treacher Collins,
Transactions of the Ophthalmological Society of the United Kingdom, **34**:18, 1914.

NEWS ITEMS

Edited by Donald J. Lyle, M.D.
411 Oak Street, Cincinnati 10, Ohio

News items should reach the editor by the 12th of the month. For adequate publicity, notices of postgraduate courses and meetings should be received three months in advance.

Deaths

Dr. V. P. Filatov, aged 81 years, died at Odessa, U.S.S.R., during October, 1956. Dr. Filatov, director of the Ukranian Research Experimental Institute for eye disease and tissue therapy, was a leading figure in soviet medical research.

Announcements

Research Study Club

The Research Study Club of Los Angeles announces its 26th annual midwinter clincial convention in ophthalmology and otolaryngology, January 14 through 25, 1957. The guest lecturers for the eye program, which will be held Monday through Friday, January 21st through 25th, will be Dr. John M. McLean and Dr. Graham Clark, New York, and Dr. Samuel J. Kimura, San Francisco. Instruction courses in ophthalmology will be conducted by Dr. Kimura, Dr. Julian N. Dow, Dr. Robert V. Shearer, and Dr. Alfred R. Robbins. A symposium will be conducted by Dr. Clarence H. Albaugh, Dr. John P. Lordan, and Mr. Russell L. Stimson, all of Los Angeles.

Further information may be obtained from:

Dr. Norman Jesberg
500 South Lucas Avenue
Los Angeles 17, California

Bielschowsky's lectures

It has been announced by Mr. Gordon Gliddon, business officer of the Baker Library, Dartmouth College, Hanover, New Hampshire, that there has been a new printing of Alfred Bielschowsky's *Lectures on Motor Anomalies*, with a foreword by Walter B. Lancaster, M.D. Three editions of these lectures have been exhausted. The contents of the present edition is said to be identical with previous issues; however, the format is slightly different. The cost of the *Lectures* is $2.50.

India offers training

The Eye Hospital, Sitapur, U.P., India, offers facilities for practical training in ophthalmology, with special emphasis on surgery, for a period of two months starting January 1, 1957. The cost is $1,000.00, which includes board and lodging provided by the hospital. Further information may be obtained from the Chief Medical Officer, Eye Hospital, Sitapur, U.P., India.

Essay contest

The Mississippi Valley Medical Society announces its annual essay contest on any subject of general medical or surgical interest, including medical economics and education. The paper must be unpublished and of interest and value to practitioners of medicine. Contributions are accepted only from physicians who are members of the American Medical Association and who are residents and citizens of the United States.

Manuscripts must not exceed 5,000 words and should be submitted in five complete copies. The winning essay will receive a cash prize of $100.00. For further details write to:

Dr. Harold Swanberg
209 W. C. U. Building
Quincy, Illinois

Research committee

A National Committee for Research in Ophthalmology and Blindness was recently formed by representatives from the four national ophthalmic societies. Members of the committee are: Dr. Bernard Becker, Washington University, and Dr. A. Edward Maumenee, The Johns Hopkins University, representing the American Academy of Ophthalmology and Otolaryngology; Dr. Michael J. Hogan, of the University of California, and Dr. Albert D. Ruedemann, Sr., Wayne University, representing the American Medical Association, Section on Ophthalmology; Dr. Alson E. Braley, University of Iowa, and Dr. David G. Cogan, Harvard University, representing the American Ophthalmological Society; and Dr. Irving H. Leopold, University of Pennsylvania, and Dr. Frank W. Newell, University of Chicago, representing the Association for Research in Ophthalmology.

The committee plans to stimulate increased interest in ophthalmic research and to co-operate with and act as an advisory group to all persons and agencies interested in blindness, the prevention of blindness, vision, and research in the basic disciplines of ophthalmology.

Wills Eye Hospital meeting

The IX annual clinical conference of the staff and Society of Ex-Residents of Wills Eye Hospital will be held at the hospital on February 8 and 9, 1957. The conference this year will be of special interest since it will be an important feature in commemorating the 125th anniversary of the founding of the hospital.

The Bedell Lecture will be delivered by Dr. John M. McLean of New York, on the subject of "Management of the primary glaucomas."

The program will also include scientific papers by members of the staff and ex-residents of the hos-

pital, technical exhibits, and a color television program sponsored by Smith, Kline, and French Laboratories which will present ocular surgery, case presentations, and demonstrations of procedures and techniques.

On Friday evening, February 8th, there will be an informal reception and supper for the ophthalmologists and their wives. On completion of the two-day program, the Wills Eye Hospital Society will hold its annual meeting and dinner at the Union League, Philadelphia, on Saturday, February 9, 1957, at 6:30 P.M.

Prior to the conference there also will be a meeting preceded by dinner, of the Ophthalmological Section of the College of Physicians in Philadelphia on Thursday evening, February 7, 1957, at which Dr. James H. Allen of New Orleans will be the guest speaker. All ophthalmologists are invited.

Ohio postgraduate course

A postgraduate course in ophthalmology will be held at the Ohio State Union Building, Ohio State University, Columbus, Ohio, March 4 and 5, 1957. Speakers will be Dr. Francis H. Adler, Philadelphia, "Diagnosis and care of muscle pareses"; Dr. George J. Hamwi, Columbus, "Clinical use of steroids"; Dr. John W. Henderson, Ann Arbor, Michigan, "Ophthalmic diagnosis of intracranial aneurysms," "Neuro-ophthalmologic syndromes," and "Differentiation of optic neuropathies"; Dr. William E. Hunt, Columbus, "Evaluation of headache"; Dr. Jay Jacoby, Columbus, "General anesthesia in eye surgery"; Dr. A. E. Maumenee, Baltimore, "Recent advances in the diagnosis and treatment of glaucoma," "Surgery of the conjunctiva and cornea," and "Diagnosis and management of uveitis"; Dr. Ted Suie, Columbus, "Practical ocular microbiology."

The registration fee for the course is $20.00. For further information write to:

Dr. William H. Havener
Department of Ophthalmology
University Hospital
Columbus, Ohio

Societies

Dallas academy

Officers of the Dallas Academy of Ophthalmology and Otolaryngology are: President, Dr. Ludwig A. Michael; vice-president, Dr. Hal W. Maxwell; secretary-treasurer, Dr. Edward A. Newell. Meetings of the society took place on October 2nd, November 6th, and December 4th. Meetings are scheduled for January 8, 1957, February 5th, February 19th, March 18th through 20th, when the Dallas Southern Clinical Society meets; April 27th through May 1st, meeting of the Texas Medical Association at Dallas; May 3rd and June 4th.

Pan-American Association

The fourth interim congress of the Pan-American Association of Ophthalmology will be held at the Hotel Statler, New York, from April 7 through 10, 1957. It will be a joint session with the National Society for the Prevention of Blindness.

At the opening meeting the Hon. Robert F. Wagner, mayor of the city of New York and honorary president of the congress, will speak, as will Dr. Frank B. Berry, assistant secretary of defense for medical affairs, and Maj. Gen. Dan C. Ogle, surgeon general, U. S. Air Force.

Under the chairmanship of Dr. John M. McLean, New York, the following symposia will be conducted: I. Diseases of the ocular fundus, Dr. George N. Wise, moderator; II. Ophthalmic surgery, Dr. John H. Dunnington, moderator; III. Therapeutics in present-day ophthalmology, Dr. Irving H. Leopold, moderator.

Dr. J. Gordon Cole is chairman of surgical clinics; Dr. Wendell L. Hughes, motion pictures; exhibits, Dr. Gerald Fonda; social activities, Dr. Elizabeth Constantine; public relations, Dr. Frank H. Constantine, chairman, and Miss Lois Stice, consultant; registration and housing, Dr. Girolamo Bonaccolto. Dr. Moacyr Alvaro is past president and executive director of the Pan-American Association, and Dr. Brittain F. Payne, New York, is president of the association and general chairman of the congress. Dr. J. Wesley McKinney, Exchange Building, Memphis 3, Tennessee, is executive secretary for North of Panama.

United Kingdom meeting

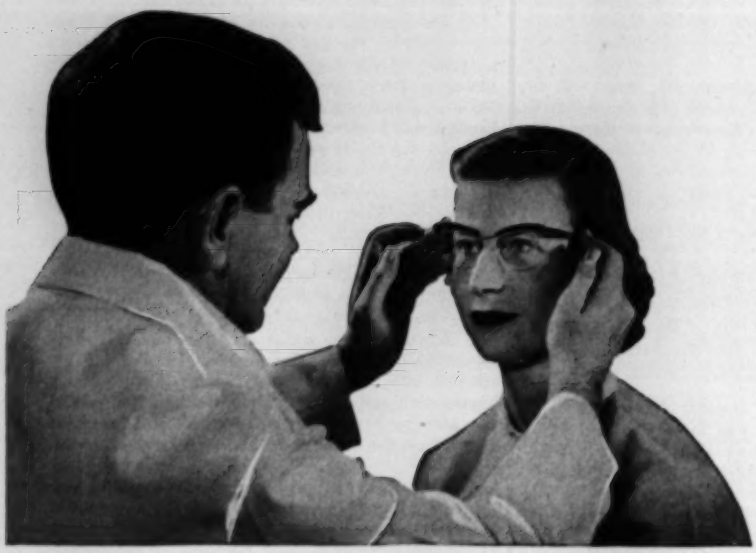
The annual congress of the Ophthalmological Society of the United Kingdom will be held at the Royal Society of Medicine, 1 Wimpole Street, London, W. 1, on April 11, 12, and 13, 1957. The presidential address will be delivered by Mr. J. J. Healy on "The intraocular circulation in arteriosclerosis and high blood pressure." The subject for discussion will be "Ocular aspects of diabetes." This will be opened by Prof. G. I. Scott, Dr. Norman Ashton, and Dr. J. D. N. Nabarro. There will also be a short symposium on "Congenital cataract." Sir Stewart Duke-Elder will deliver the Bowman Lecture, his subject being "The etiology of glaucoma."

Personals

The Section on Ophthalmology, American Medical Association, awarded a $250 prize to Dr. T. E. Sanders and Dr. Albert P. Ley of Saint Louis, for their paper entitled, "Fungus keratitis," judged on the basis of presentation and originality. This award was made at the Section meeting in Chicago in June, 1956.

The Lister Medal of the Royal College of Surgeons (England) for 1956 has been awarded to Sir Stewart Duke-Elder, who will deliver the Lister Lecture in London on April 5, 1958, under the auspices of the Royal College of Surgeons of England.

Dr. Edwin B. Dunphy, Boston, will deliver the Sanford R. Gifford Lecture on February 15, 1957, during the annual clinical meeting of the Chicago Ophthalmological Society.



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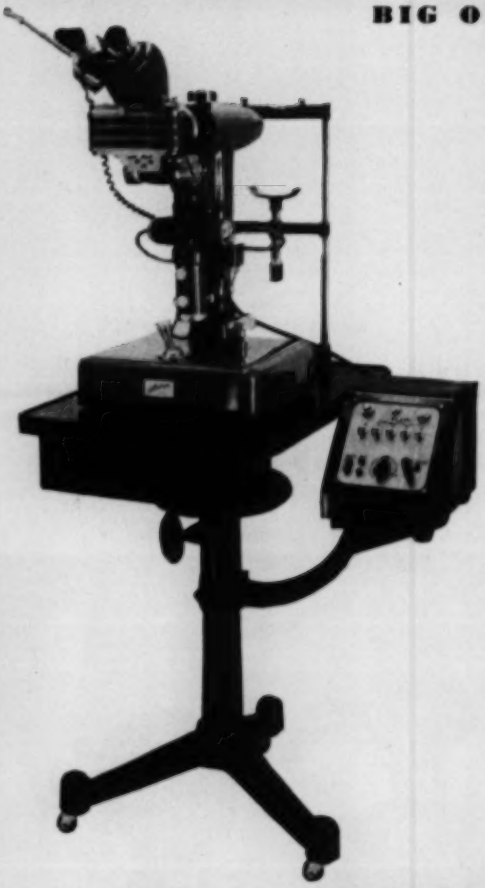

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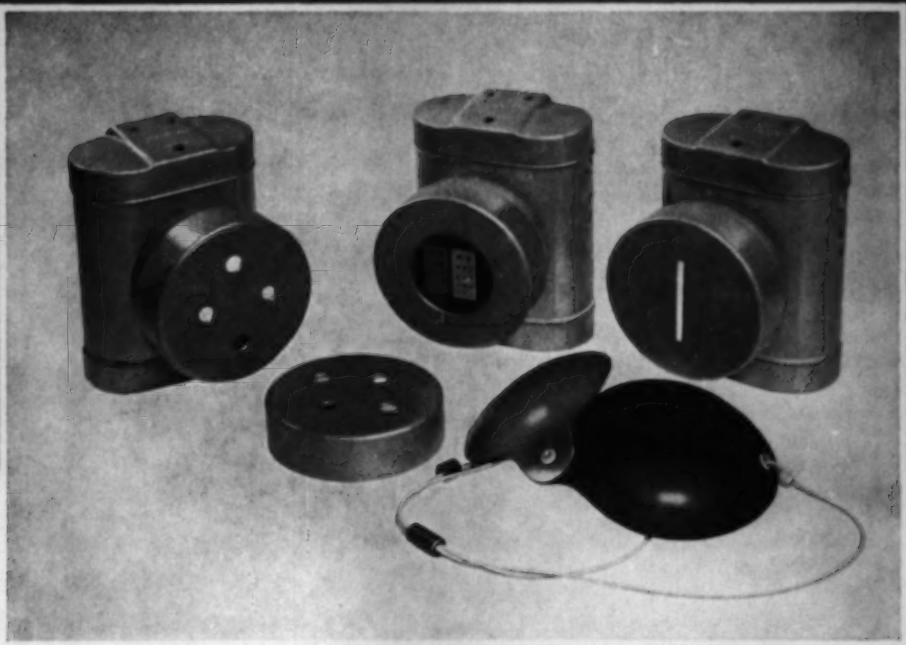

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